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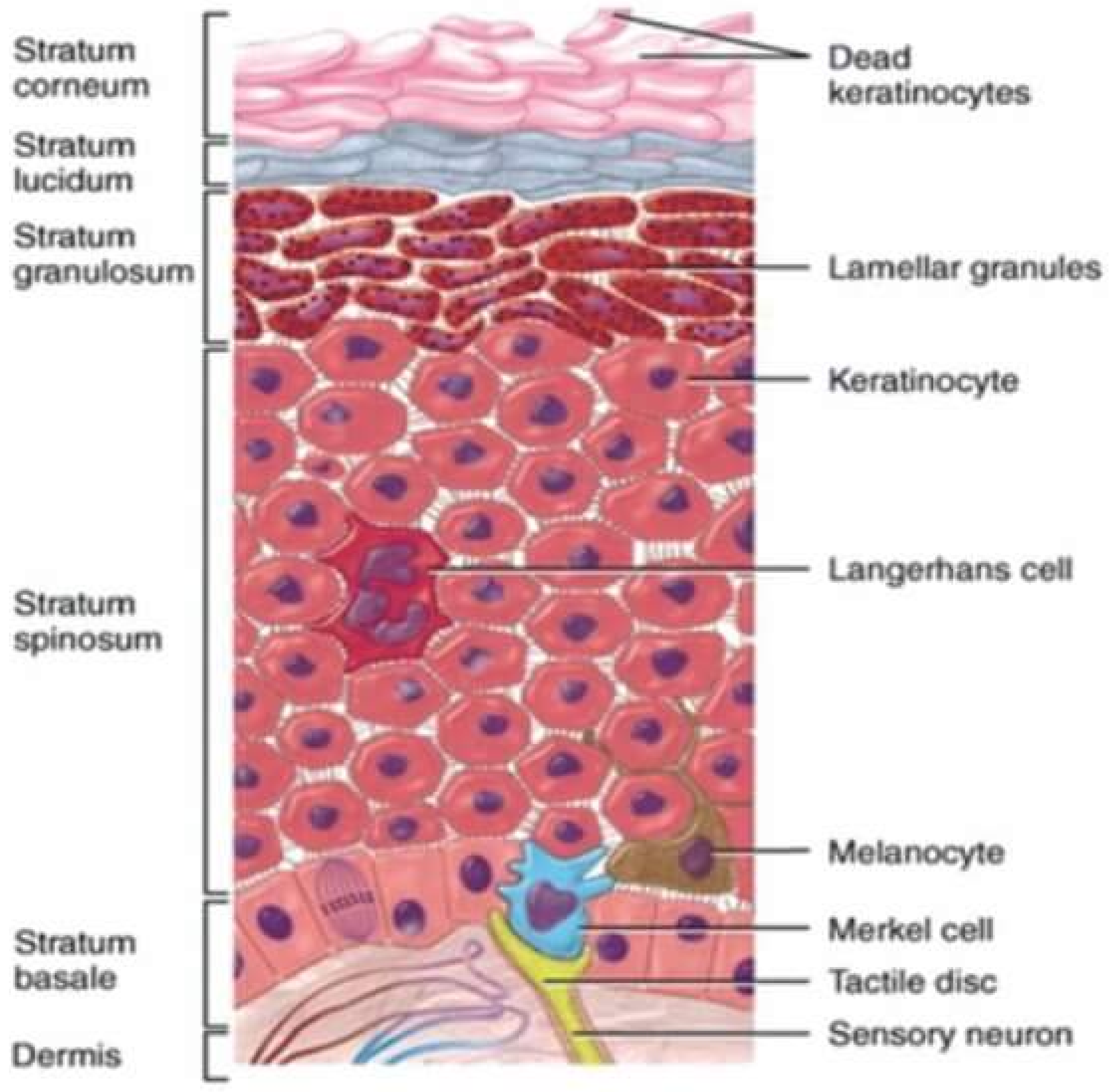
Cells of Epidermis

Introduction of skin & its appendages

Skin
- Epidermis – Dermo epidermal junction (DEJ) or Basement membrane zone (BMZ) present between dermis & epidermis.
- Dermis
- Subcutis



Skin can be divided into various reactive units

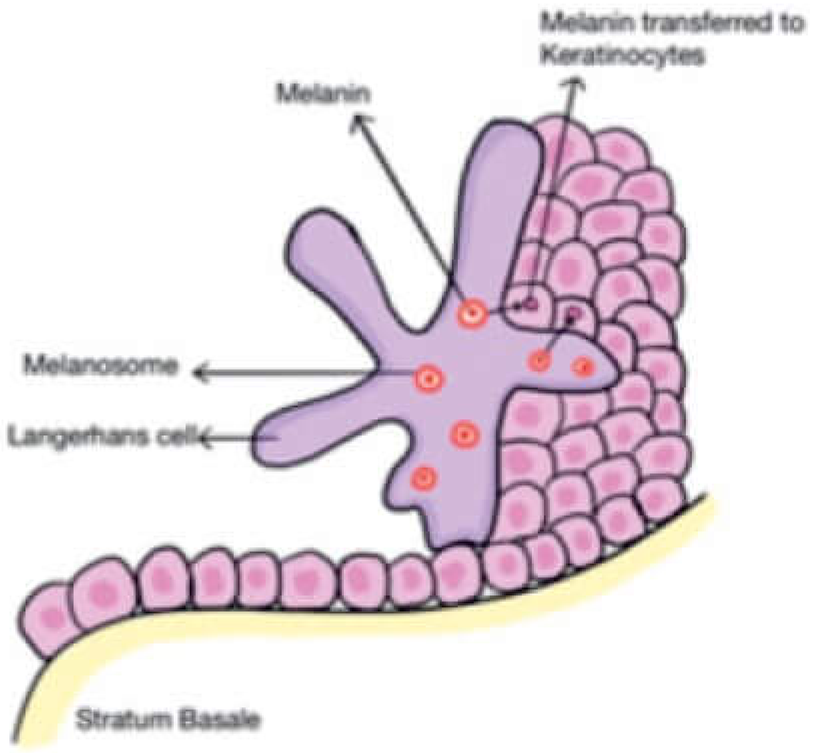
Reactive units – Pathophysiological unit of skin

1. **Superficial reactive unit**
   - I) Epidermis
   - ii) DEJ / BMZ
   - iii) Papillary dermis
   - iv) Superficial vascular plexus
2. **Deep reactive unit**
   - i) Reticular dermis
   - ii) Deep vascular plexus
3. **Subcutaneous reactive unit**
   - i) Subcutaneous layer
   - ii) Appendages
     - Hair
     - Nails
     - Glands
       - Sweat gland
       - Sebaceous gland

Epidermis

Cells of epidermis

1. Keratinocytes (90%)
2. Melanocytes
3. Merkel cells
4. Langerhans cells



I) Keratinocytes:

Hallmark
↓
Presence of keratin filament
Or
Tonofilaments
Or
Intermediate filament

Keratin

Soft
(decrease disulphide bond)
eg. Skin

Hard
(increase disulphide bond)
eg. Nails

→ Keratin Heterodimers
In stratum basale
↓
K5, K14 keratin filaments (present)

K5
K14
} Both join to form heterodimer

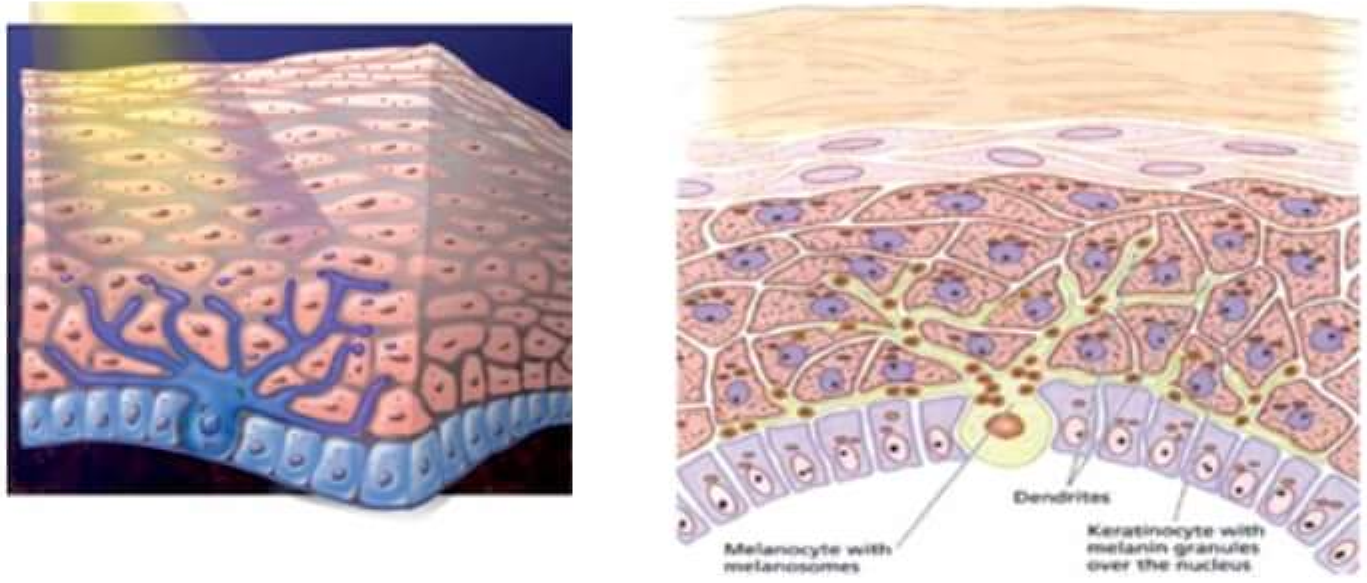
Layers Of Epidermis

1. Stratum corneum
2. Stratum lucidum
3. Stratum granulosum
4. Stratum spinosum
5. Stratum basale

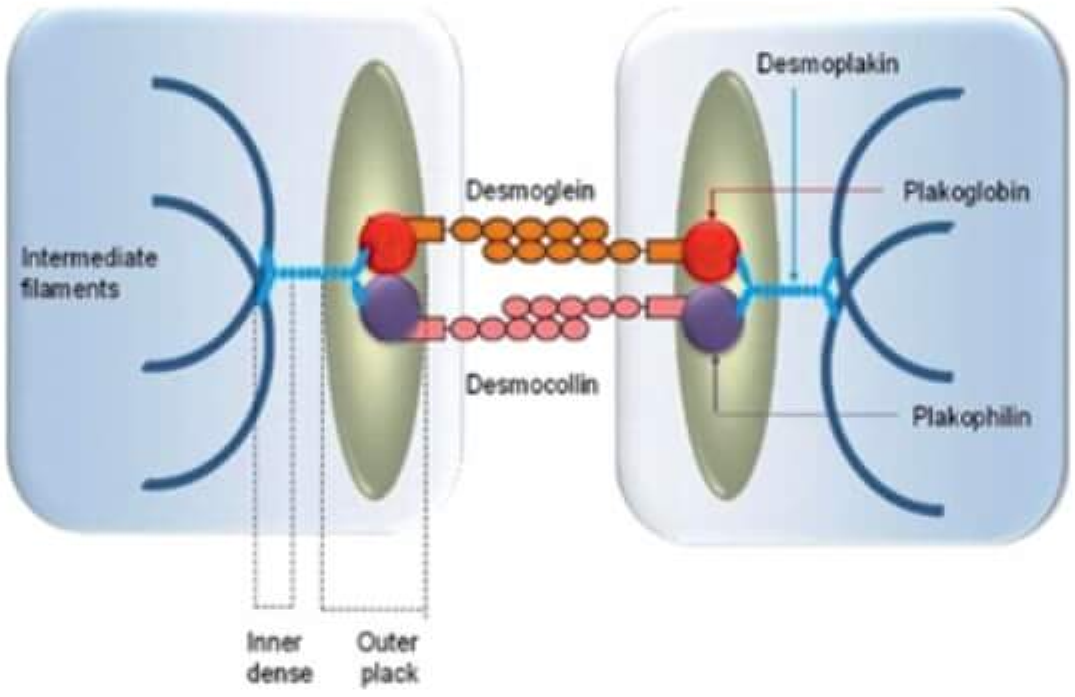
Keratin heterodimer		**sites**	
1. K 5/14	→	Stratum basale	→ If defective Epidermolysis bullosa simplex
2. K 1/10	→	Stratum spinosum	
3. K 2/11	→	Stratum granulosum	
4. K 3/12	→	Cornea	→ If defective → Corneal dystrophy
5. K 4/13	→	Buccal mucosa	
6. K 6/16	→	Nails	
7. K 8/18	→	Liver	→ If defective → Liver cirrhosis

II) Melanocytes

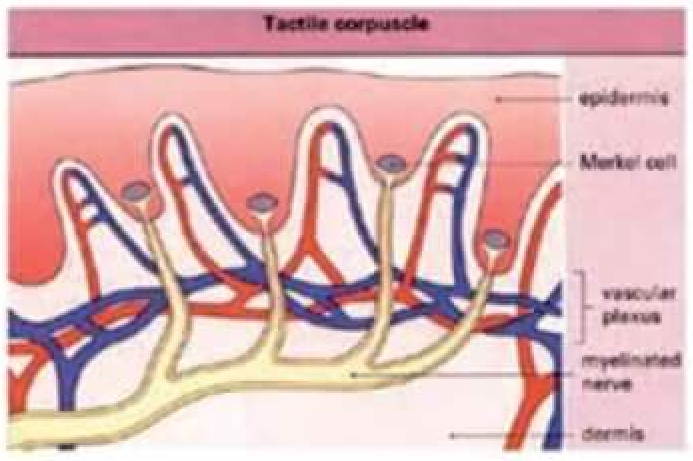
→ Are dendritic cell present in Stratum basale


Dendrites
Melanocyte with melanosomes
Keratinocyte with melanin granules over the nucleus

Melanocytes



Desmosome: showing arrangement of proteins



Merkel cell

Melanocytes → Synthesize Melanosomes
↓
(Melanin containing cell organelles)

Melanosomes are transferred to keratinocytes via dendrites.

36 keratinocytes / melanocytes → forms Epidermal melanin units (EMU)

EMU → Gives uniform skin colour

Different shades of skin colour can be due to:

I) Difference in number, size & distribution of melanosomes
ii) Diffuse in proportion of Eumelanin and pheomelanin
Number of melanocytes / cm^2 Equal in all individuals

Melanogenesis

Tyrosine
↓ Tyrosinase
DOPA

} Rate limiting step

↓
DOPA Quinone

Dopa chrome → Eumelanin (True) → imparts Brown / black

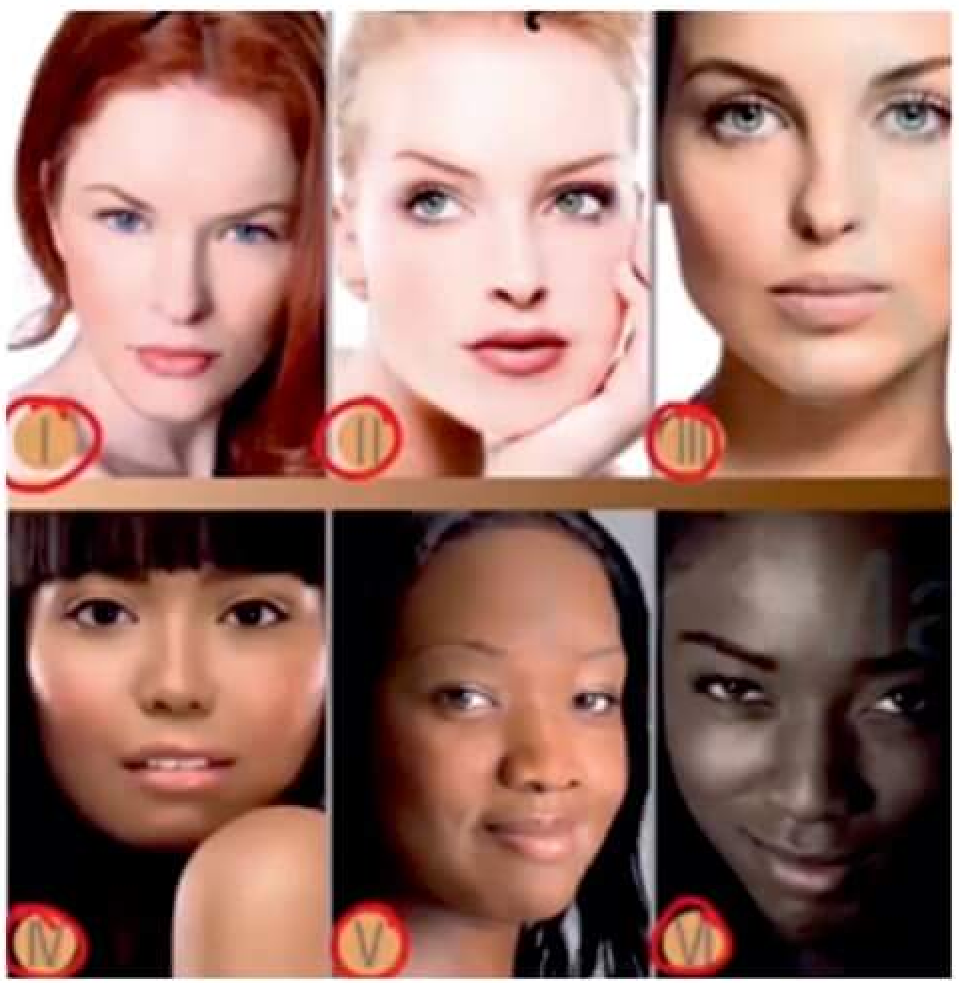
Cysteinyl DOPA → Pheomelanin (False) → imparts Red / yellow

Q The father of modern Academic dermatology

Thomas B. Fitzpatrick

Thomas B. Fitzpatrick
↓
He divided skin into 6 types
→ known as Fitzpatrick classification

This classification based on property of skin to tan or burn.



Increased Pheomelanin

Increased Eumelanin

Type I- Never tan, always burns
Type VI- Always tan, never burns

Pigmentary Disorders

Melanocytes

1. **Vitiligo**
[due to absence of Melanocytes]
[depigmented Lesions]
→ Autoimmune

2. **Piebaldism**

[due to absence of Melanocytes]

[depigmented Lesions]

→ Genetic

→ Characterized byLocalised patch over forehead

→ White forelocks

→ Areas of pigmentation in between

3. **Lentigens**

→ Increased localised no. of melanocytes

→ Hyperpigmented macules (neither raised nor depressed, only change in colour)

→ < 0.5cm

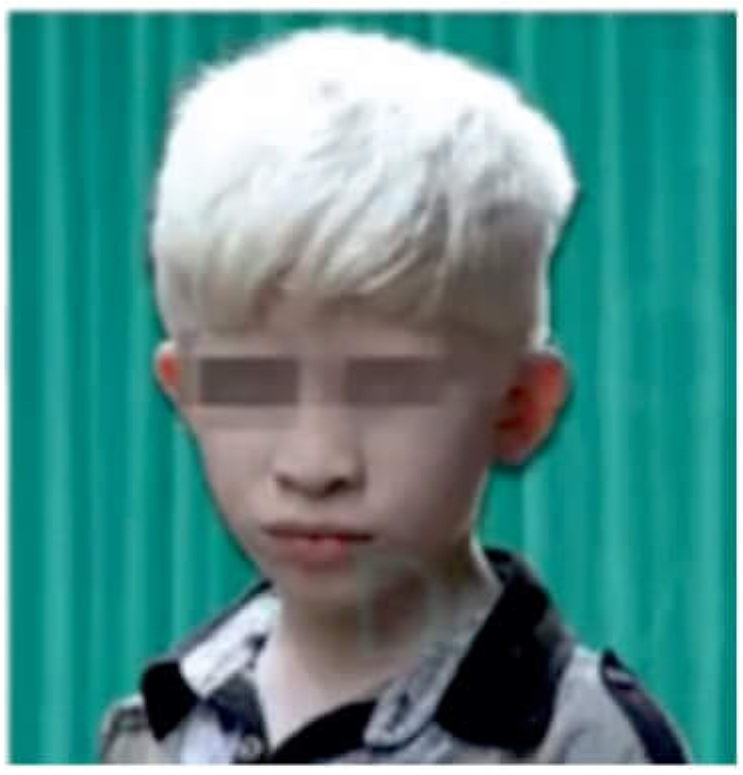
Melanosomes

1. **Nevus acromicus or Nevus depigmentosus**

- due to defect in transfer of melanosomes.

On Diascopy: Lesion can be differentiated from surrounding skin.

Melanogenesis



1) **Melasma**

→ due to increase in melanogenesis

2) **Freckles**

→ due to increase in localized melanogenesis

→ Hyperpigmented macules

3) **Albinism (Occulo - cutaneous disorder)**

Occulo] - Ocular: Photophobia
Cutaneous] (defective Melanogenesis) depigmented skin & hair
Disorder]

→ Inheritance-Autosomal recessive, genetic mutation in TYR (tyrosinase gene)

→ Defect- Deficiency of melanin synthesis but melanocytes are normally present and distributed.

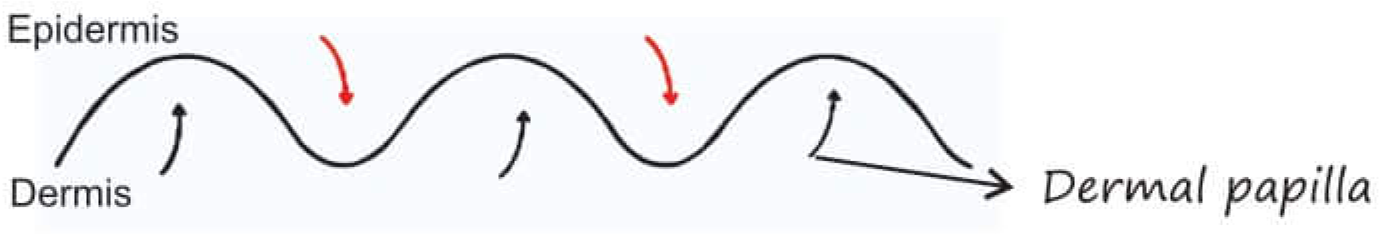
→ Clinical feature

- Generalised depigmentation of skin, hair and eye (iris/eye) due to loss of melanin production.
- Associated with photophobia, Nystagmus and increased risk of skin malignancies (squamous cell carcinoma).

III) Merkel cells or Touch cells

→ Type I, slow adapting touch receptors

→ Present mainly in stratum basale



Transverse section of skin

Q. Merkel cells are present in?

a. Dermal Papillae
b. Papillary dermis
c. Rete ridges
d. Epidermis

Ans. C. Rete ridges (are projection of lower epidermis)

Touch receptors

Type I/ slow	**Type II/Rapid**
Eg.	Eg.
1. Merkel cells	1. Hair end organs
2. Ruffine's nerve endings	2. Meissner corpuscle
3. Free nerve endings	3. Pacinian corpuscle

Q. Vibration is carried by Pacinian corpuscles.

IV) Langerhans cells:

→ They are Antigen presenting cells that help in Phagocytosis
→ Dendritic cells present in → Stratum spinosum Q
→ Characteristic: Rod/racquet granules
↓
Birbeck granules help in → Receptor mediated Endocytosis.

On Immunohistology

These cells are (+)ve CD_{1a}, CD_{207}, S_{100} (CD_{207} also known as Langerin)
(-)ve CD_{14}, CD_{68}, CD_{163}

→ Decrease langerhan cells in 1. Psoriasis
2. Sarcoidosis
3. Contact dermatitis

→ Increase Langerhans cells - Langerhans cells Histiocytosis (or) Histiocytosis - x

Langerhans cells Histiocytosis

Subtypes	Age of onset	clinical features
1. Eosinophilic Granuloma	<8 years	- Localised to bones - MC bone involved - skull (Temporoparietal area) (Lesion-lytic)
2. Hand Schuller Christian disease	< 5 years	Triad - Multiple bone defects - Diabetes Insipidus - Exophthalmus
3. Letterer Siwe disease	<2 years ↓ (decreasing order)	- Generalised/ Acute fulminant Condition ↓ (increase in c/f) 80% - Bone 50% - skin 30% - Hepatosplenomegaly Lymphadenopathy

Skin manifestation of Langerhans cells histiocytosis
↓
Seborrheic Dermatitis

4. Hashimoto Pritzker disease
→ subtype of Langerhans cell histiocytosis
→ present since birth
→ rare
→ self-limiting condition

Important fact-

*PeutzJegher Syndrome

↙ Autosomal Dominant

↓ GI Polyps
MC - Jejunal Hamartoma

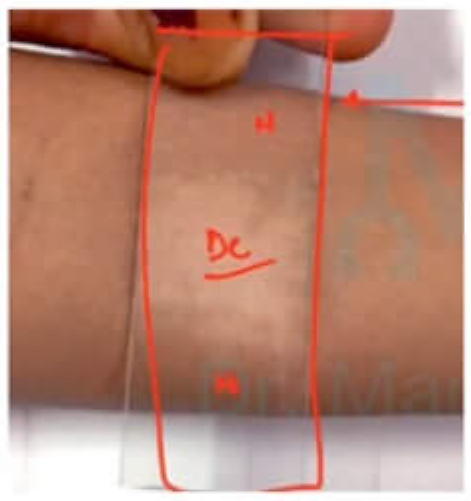
↘ Lentigen
Site - perioral (Lips)
Buccal mucosa

* **Freckles/ Ephelids**

Normal in skin type - I, II
Disorder - Xeroderma Pigmentosa
- DNA repair defect
- [Nucleotide excision defect]
- In children - squamous cell carcinoma

* **Nevus Anemicus**

defect → catecholamine induced vasoconstriction
↓
pale skin

Nevus Acromicus	Nevus Anemicus
1) Diascopy: Margin differentiated From surrounding skin.  - Hemangioma / Purpura	1) Diascopy: Margin merge with Surrounding skin.
2) Heat: Erythema develops	2) Heat: Erythema does not develop

Cells of Epidermis	Derived from
1) Keratinocytes	Ectoderm
2) Melanocyte	Neural crest
3) Merkel cells	Ectoderm> Neural crest
4) Langerhans cells	Bone marrow (mesoderm)

Lesions follow Seborrheic distribution

↓

i) Scalp
ii) Retro auricular area
iii) T- Zone (face)
iv) Nasolabial fold
v) Pre- sternal
vi) Inter scapular

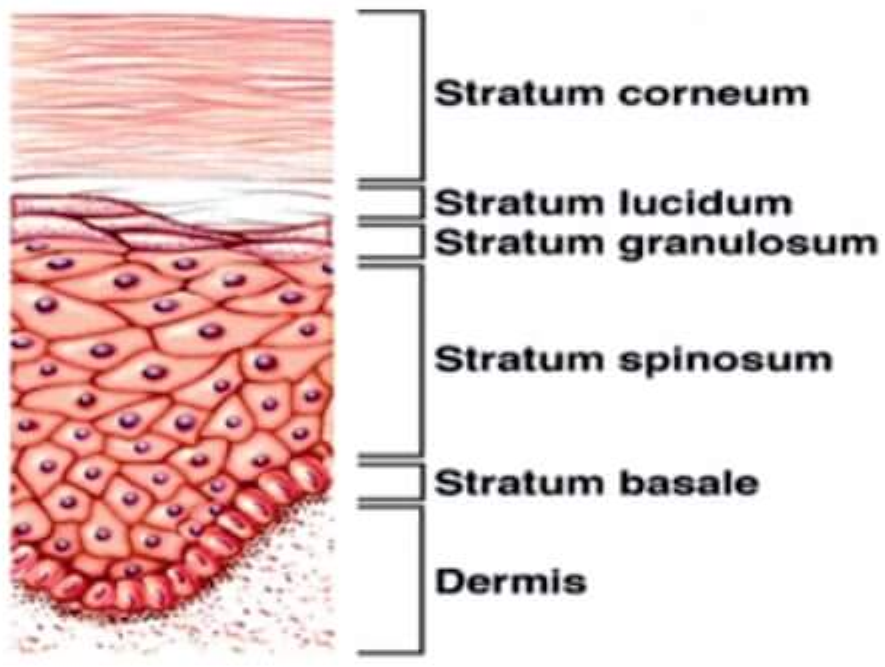
Lesions following Seborrheic Distribution :

1) Langerhans cell histiocytosis
2) Pemphigus foliaceous
3) Seborrheic Dermatitis
4) Darier's disease

LAYERS OF EPIDERMIS

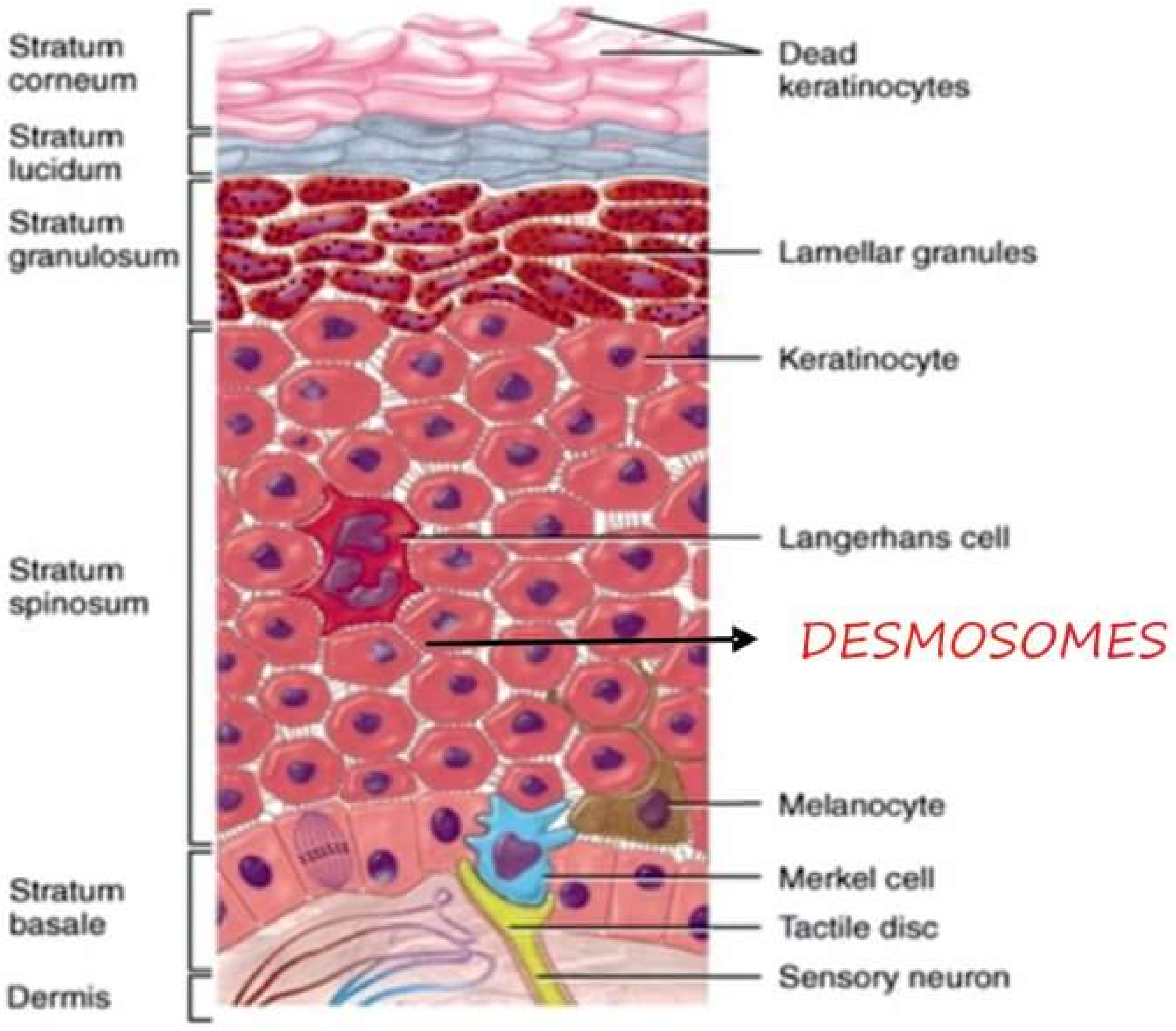
- Stratum Basale is formed 1st (below upwards)
- Followed by stratum spinosum → granulosum → lucidum → corneum
- In preterm / low birth weight baby – **stratum corneum is deficient**

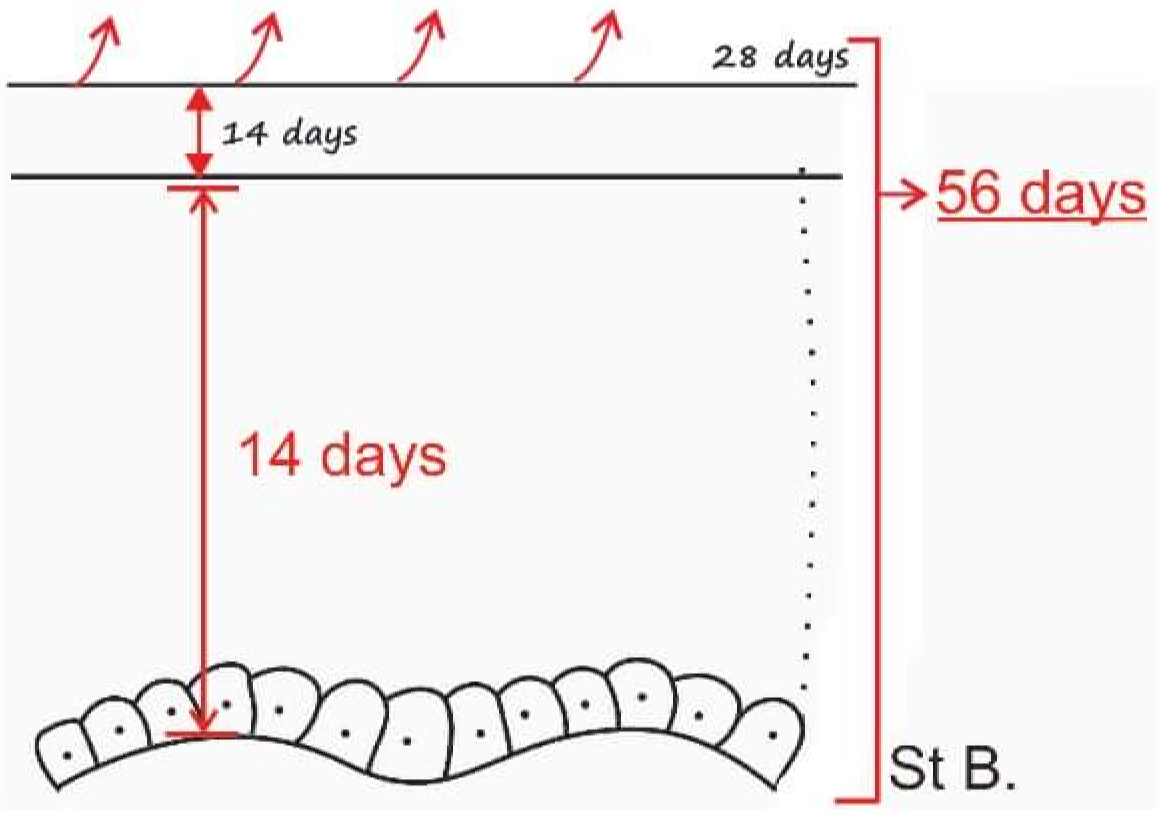
Layers of epidermis:
Le G S



1. Stratum Basale – single cell layer

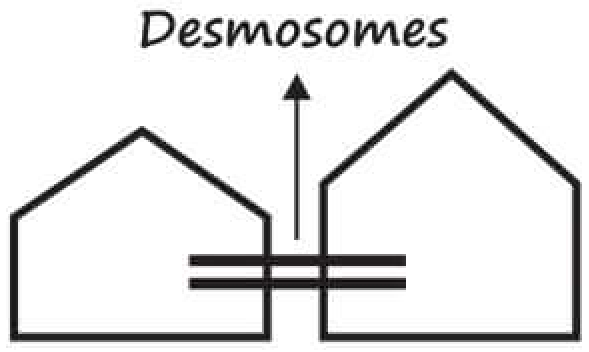
- Highly mitotically active keratinocytes having housekeeping organelles
- **Epidermal turnover time – skin doubling time**
  - → Time taken by keratinocytes to move from St. Basale to environment
  - → Around 14 days are required to move from Stratum Basale & to reach lowermost Layer of stratum corneum & 14 days to Cross stratum corneum
  - → Sloughing off requires 28 days
    So total ETT= 56days
    In Psoriasis ETT is reduced to 4 days





2. Stratum Spinosum – Prickle Cell Layer

- Spines (Desmosomes) prominent here
- Thickest Layer



Desmosomal protein / Ca^{2+} dependent Adhesion molecules

Gene family

1. Cadherin
2. Plakin
3. Armadilo

Eg-

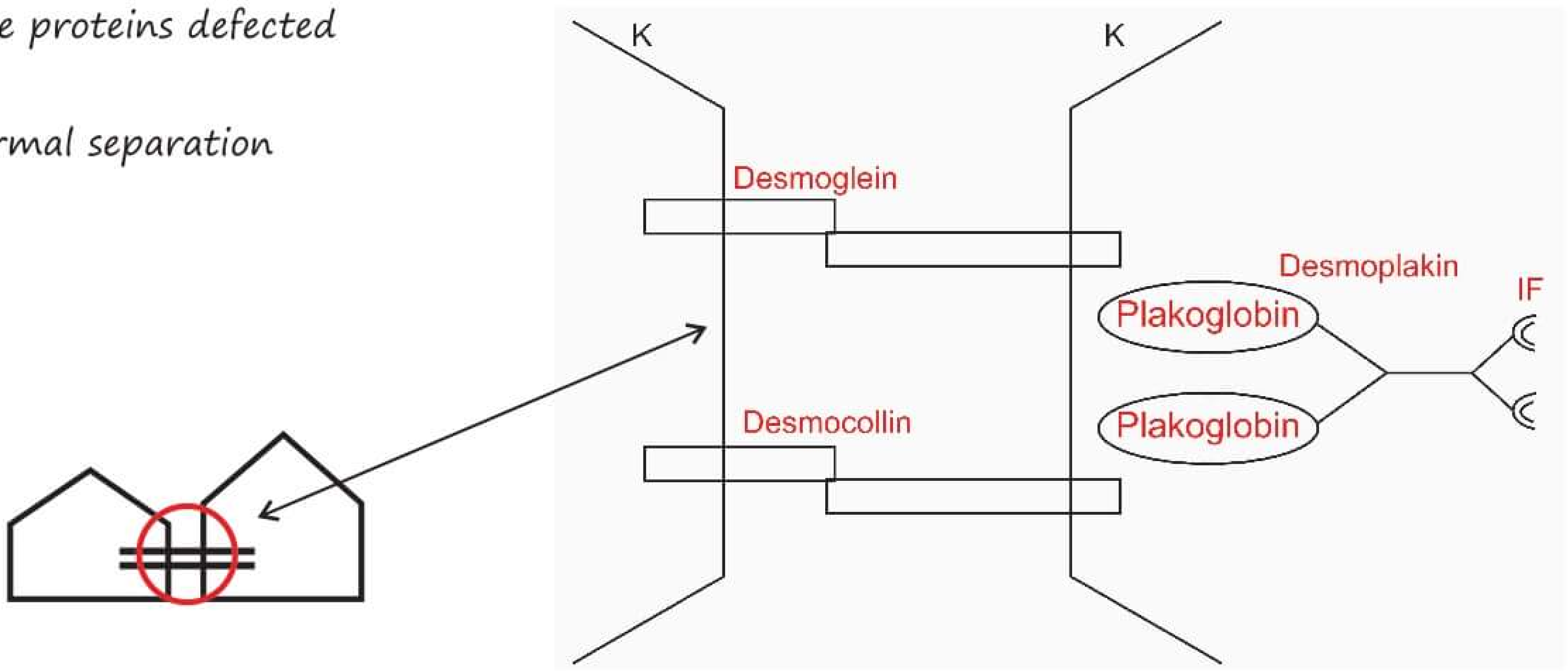
1. Desmoglein (Dsg)
2. Desmocollin (Dsc)
3. Desmoplakin (DP)
4. Plakoglobin (Pg)
5. Plakophilin (PP)

} Defect lead to intraepidermal disorder

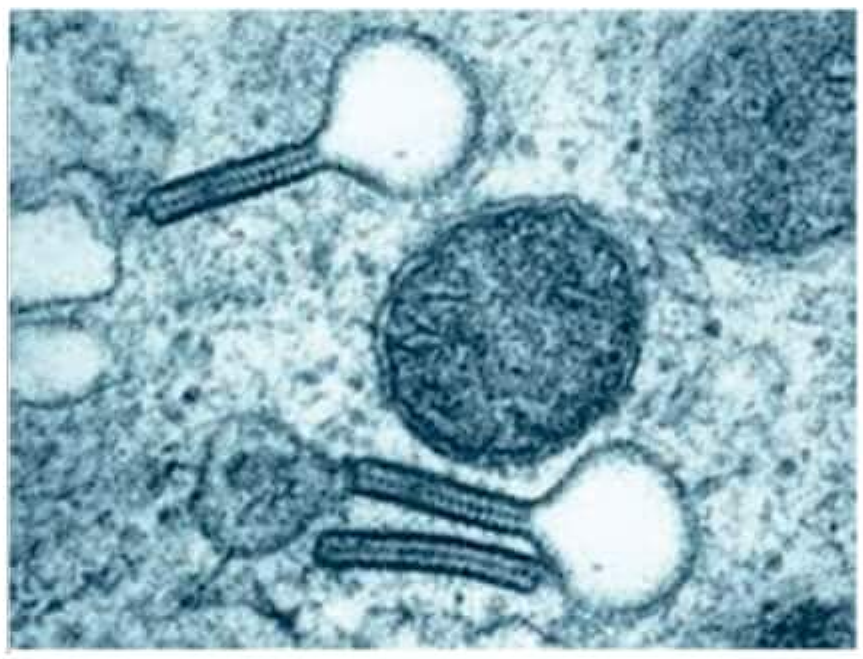
- Desmoglein – in pemphigus (intraepidermal blistering disorder)
- Any of these proteins defected

  ↓

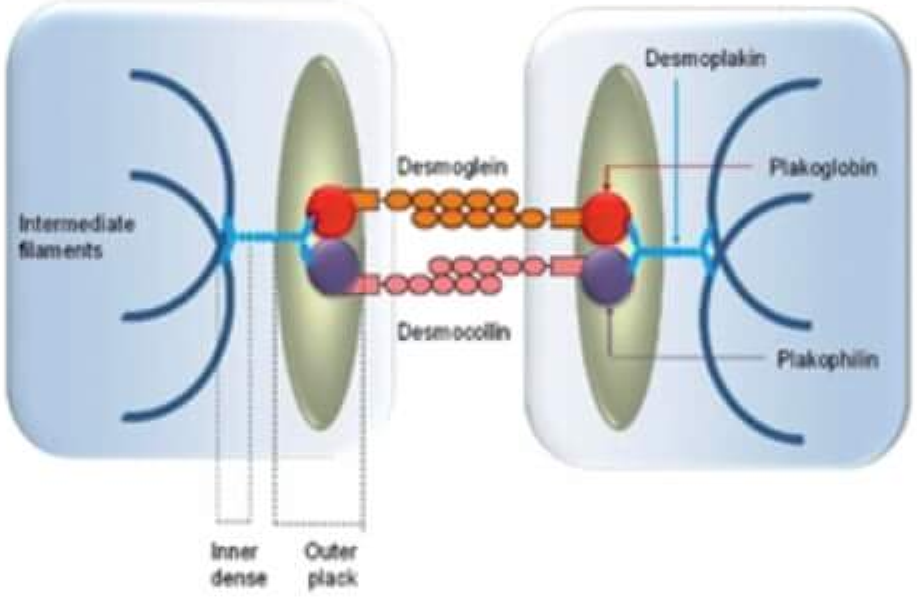
  Intra epidermal separation



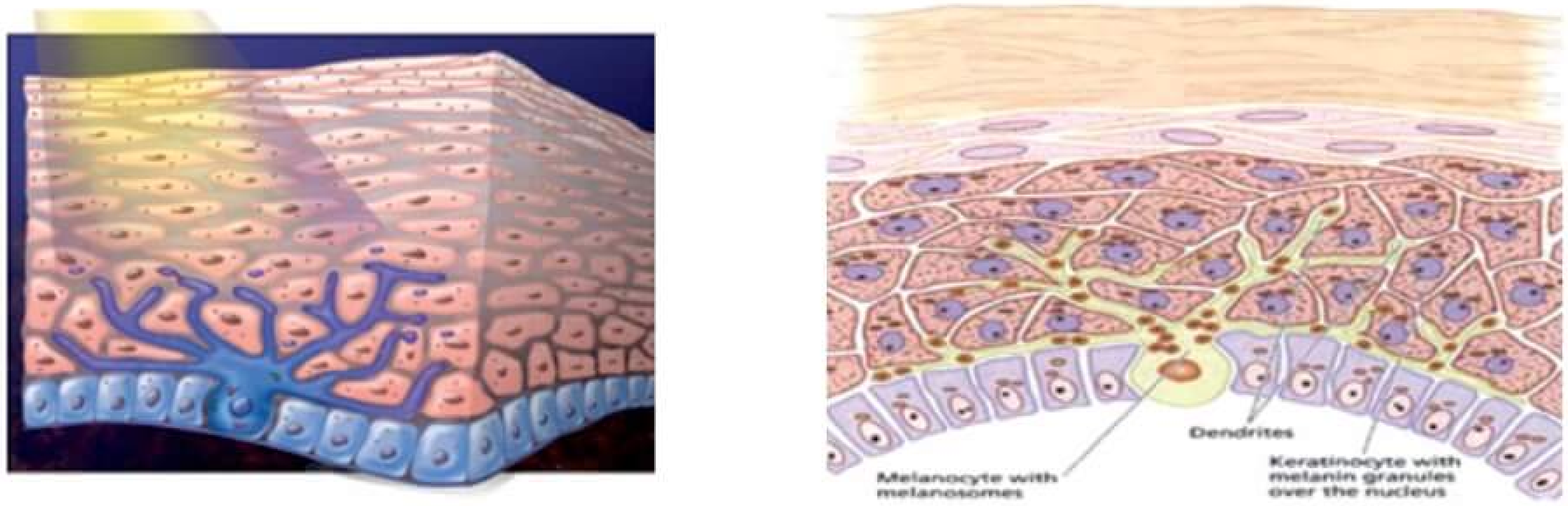
Arrangement of desmosomal proteins within a desmosome



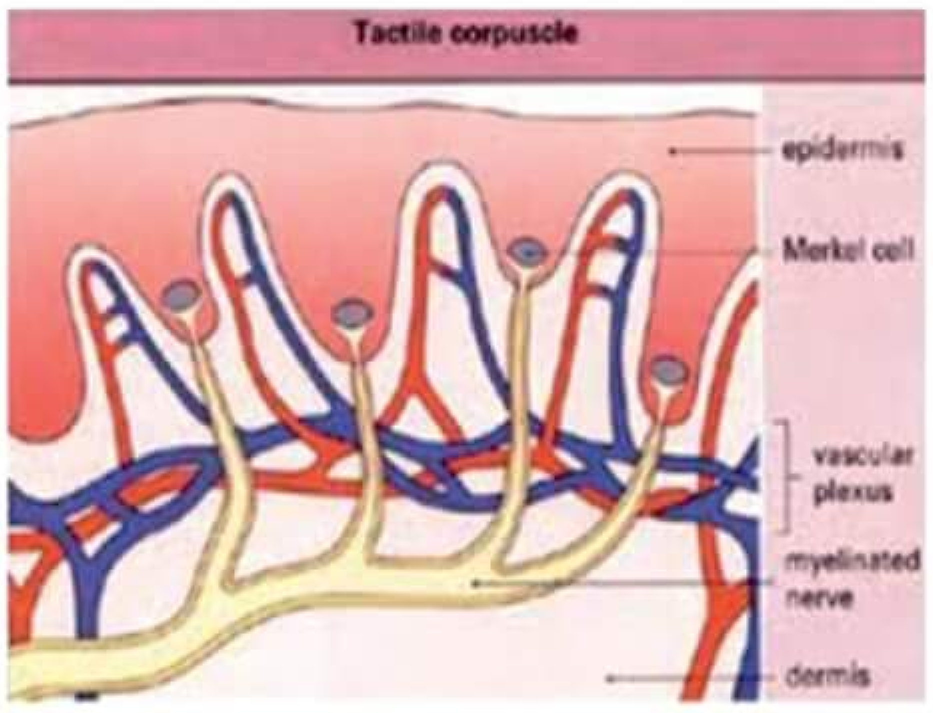
Birbeck granules in Langerhan cells helps in Receptor mediated endocytosis



Desmosome: showing arrangement of proteins


Dendrites
Melanocyte with melanosomes
Keratinocyte with melanin granules over the nucleus

Melanocytes



Merkel cells

3. Stratum Granulosum

- Granular Cell Layer
  1. **Keratohyalin granule** –Pre-filaggrin
     ↓
     Precursor molecule
     – helps in filament aggregation
  2. **Membrane coating granule**
     Aka – Lamellar granule / Odland bodies
     ↓
     Lipid
     ↓
     Protective Barrier

Important Facts

I. Odland Bodies – present in
→ Upper part of stratum spinosum
→ Type II alveolar cells in lungs (surfactant)

II. Ichthyosis vulgaris
Ichthyo → Fish like scales
Vulgaris → most common
– AD
– Deficiency of Filaggrin

III. Netherton syndrome
1) Atopic dermatitis
2) Ichthyosis Linearis circumflexa
3) Bamboo hair (Trichorexhis invaginata)

4. Stratum Lucidum

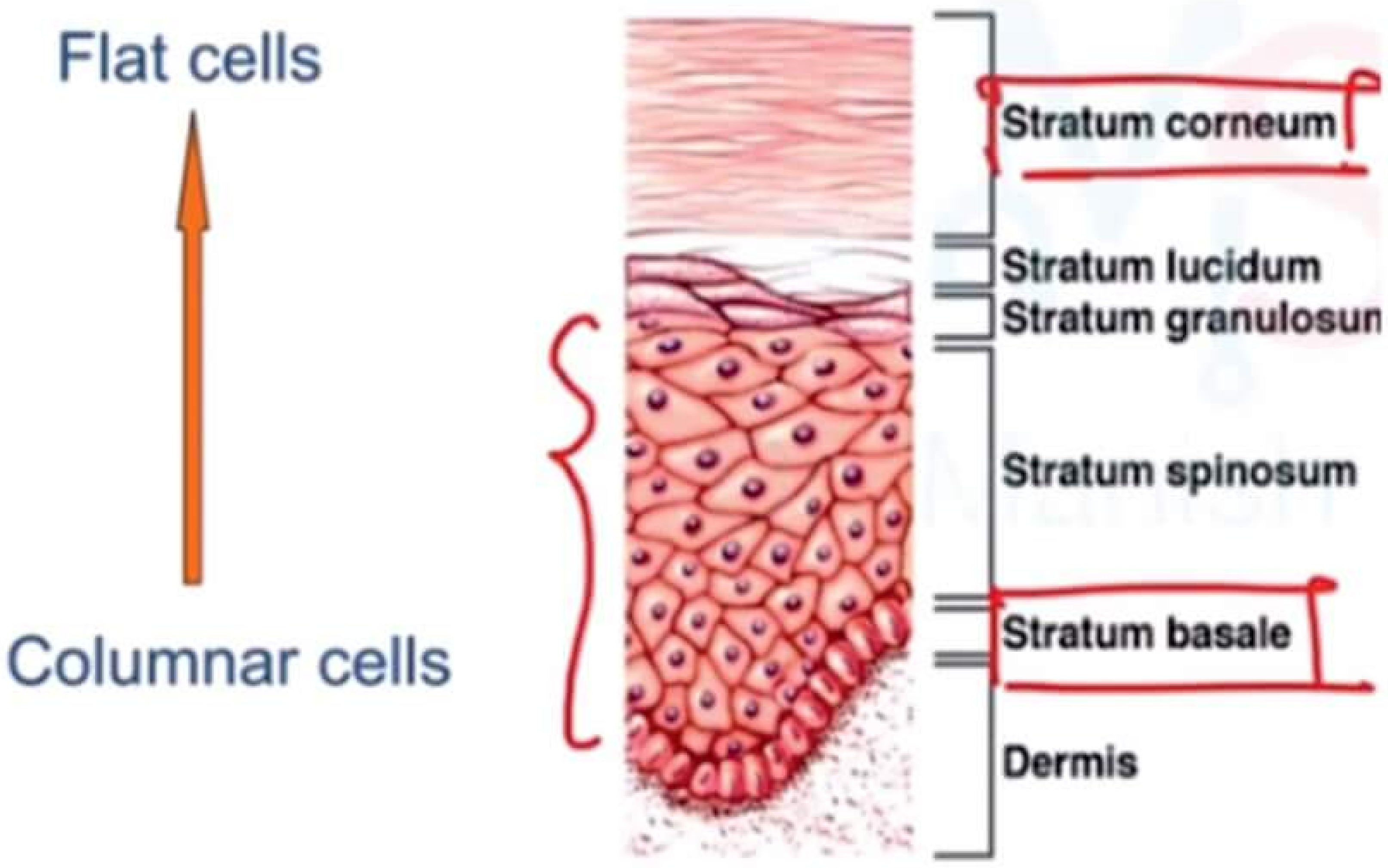
- Aka clear cell layer
- Translucent due to presence of refractile granules of Eleidin
- Present in palm & Soles

5. Stratum Corneum

- Dead keratinocytes present
- Lack Nucleus
- Stratum Corneum

  ↑ 1. Flat Cells
  2. ↑ Size
  3. Loss of nucleus
  4. Loss of moisture (Dehydrated)
  5. Dehydrated cells

  Stratum Basale



HISTOPATHOLOGICAL FINDING

1. PARAKERATOSIS

Def. → Retention of nucleus in stratum corneum

Eg → PEA'S$_2$
- → Psoriasis
- → Eczema
- → Actinic Keratosis (A.K)
- → Seborrheic Dermatitis
- → Squamous Cell Carcinoma

Important Facts

Actinic Keratosis

- → Premalignant condition
- → Seen in sun exposed areas
- → Scaly lesions / Keratotic lesions
- → Treatment
  - I. Topical 5 Fluorouracil & imiquimod
    ↓ MOA
    [Toll LR- 7, 8 agonist]
  - ii. Photodynamic therapy

AIIMS Q: What is the color change seen in lesion after photodynamic therapy ?

Explanation : Aminolevolunic acid is applied over lesions
↓
Exposed to laser light
↓
Converted to Protoporphyrin (Red) IX
↓
Cytotoxic for premalignant cells

2. DYSKERATOSIS

Def. → Premature or abnormal Keratinization

E.g → 1. Malignant Condition → Squamous Cell Carcinoma
2. Premalignant → Bowen's disease, Paget's Disease
3. Benign → Hailey – Hailey disease
→ Darier's disease → (Most important e.g)

Important facts

darier's disease

Corps grains- upper epidermis
Corps ronds- lower epidermis

3. SPONGIOSIS

Def → Extracellular accumulation of fluid in stratum spinosum

E.g → Acute eczema that is oozing out / boiling out

4. BALLOONING

Def → Intracellular accumulation of fluid in stratum spinosum

E.g → Acute eczema

5. ACANTHOSIS

Def → Thickening of stratum spinosum

E.g → Chronic eczema (Lichenified lesions)

NEET 18 Ques – Itchy lichenified lesions present over flexors of elbows & Knees is seen in ?

Ans → Atopic dermatitis ie chronic eczema

Lichenified lesions characteristic feature mnemonic "HMP"

1. Skin thickening – Hypertrophy
2. ↑ Surface markings
3. Pigmentary changes

Important Facts

*Poikiloderma mnemonic "ATP"

1. Skin atrophy
2. Telengeictasia
3. Pigmentary changes

This condition is seen in Mycosis fungoides

↓

(CTCL) Cutaneous T-Cell Lymphoma

*Acanthosis types

1) Regular – eg. Chronic eczema, psoriasis
2) Irregular – eg. Lichen planus

6) HYPERGRANULOSIS

Definition – Thickening of stratum granulosum eg. Lichen planus (Wedge shaped).

7) ANGRANULOSIS / HYPOGRANULOSIS

Definition – Absence / Thinning of stratum granulosum

Eg. Psoriasis

- Ichthyosis vulgaris

Q- Absence of stratum granulosum is seen in?

Ans. Psoriasis, Ichthyosis vulgaris.

8) MICRO ABSCESS (M/A)

Definition – Small collection of cells (Any type of cell)

1. Neutrophilic M/A

Eg

i. Papillary Tip M/A – Dermatitis herpetiformis

ii. Munro's M/A – Psoriasis (Stratum corneum)

iii. KOGOJ Pustules – Psoriasis (Stratum spinosum)

2. Lymphocytic M/A

Eg.Pautner's M/A – Mycosis fungoides

3. Eosinophilic M/A

Intraepidermal eosinophilic M/A → pemphigus vegetans.

Most important microabscess

- *Munro's M/A - Psoriasis
- *Pautrier's M/A - Mycosis fungoides

* Botryomycosis (Misnomer because it is not a fungal infection)

- Caused by staph aureus
- Small collection of Neutrophils is seen - granuloma

↓

Thus can be known as microabscess.

9) Acanthlysis

Definition – Separation of keratinocytes due to defect in desmosomes.

- Cells formed after separation are called acanthocytes / acantholytic cells.

Acanthocytes → oval shaped.

Clinical features

- → Nucleus occupies 7/8th portion of cell.
- → Perinuclear halo.

Tzanck smear – Cytology test

Acanthocytes ↓	MNGC ↓	Dyskeratotic Keratinocytes ↓	Necrotic Keratinocytes ↓
Pemphigus	1) Herpes 2) Chicken Pox	Davies's disease	Epidermal necrosis ↓ SJS / TEN

E.g of Acantholysis

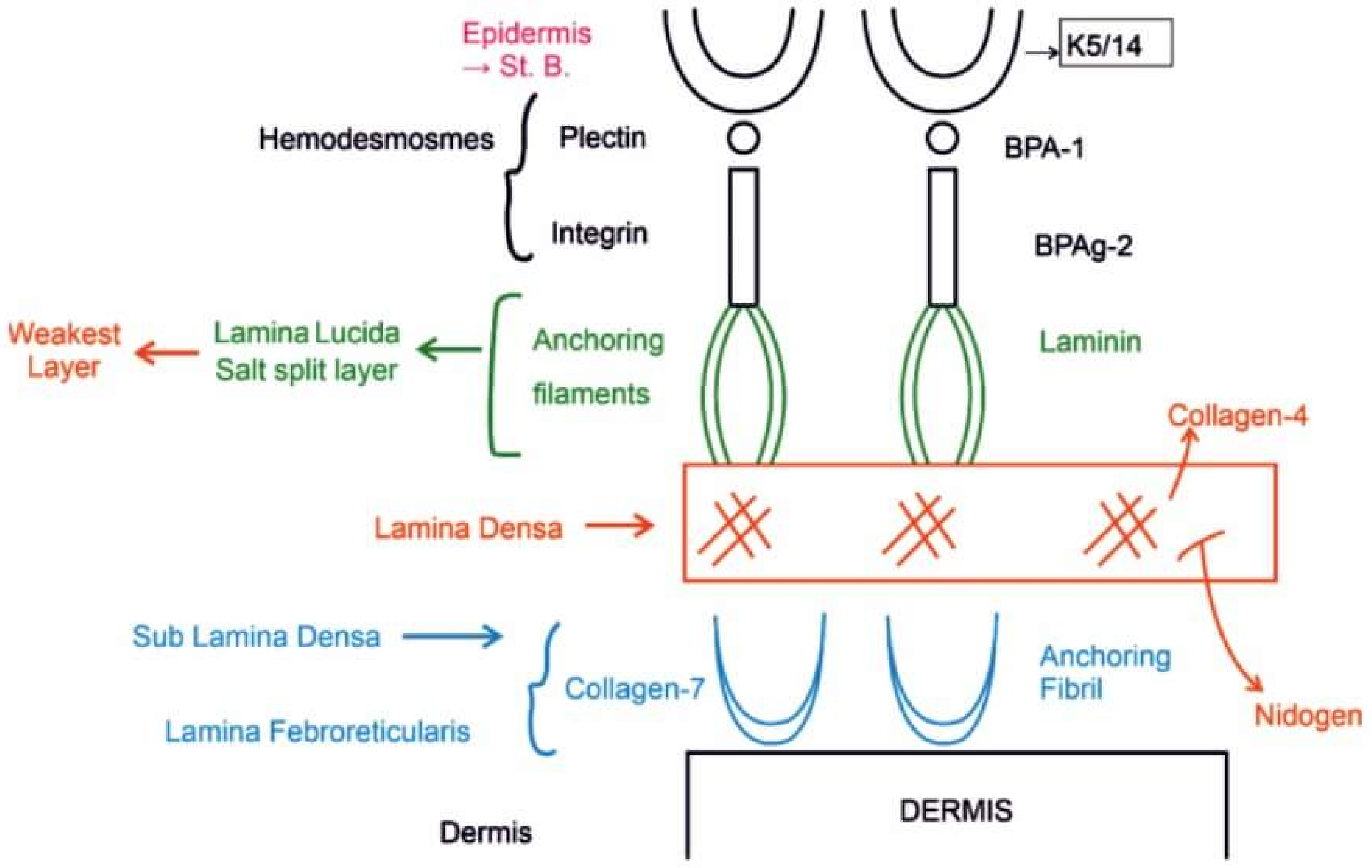
Intraepidermal Disorder

1. **Pemphigus vulgaris → because of defective Desmoglein-3>1**
2. Pemphigus foliaceus → because of defective Desmoglein-1
3. Paraneoplastic disease → because of defective plakin family- Desmoplakin, Plakoglobin, envoplakin
4. Darier's disease → because of Ca^{2+} ATPase defect
5. Hailey Hailey disease → because of defective Ca^{+2} Atpase
6. Subcorneal pustular dermatosis → because of the defective desmocollin

Sneddon - Wilkinson disease

BASEMENT MEMBRANE ZONE / DERMO-EPIDERMAL JUNCTION (DEJ)

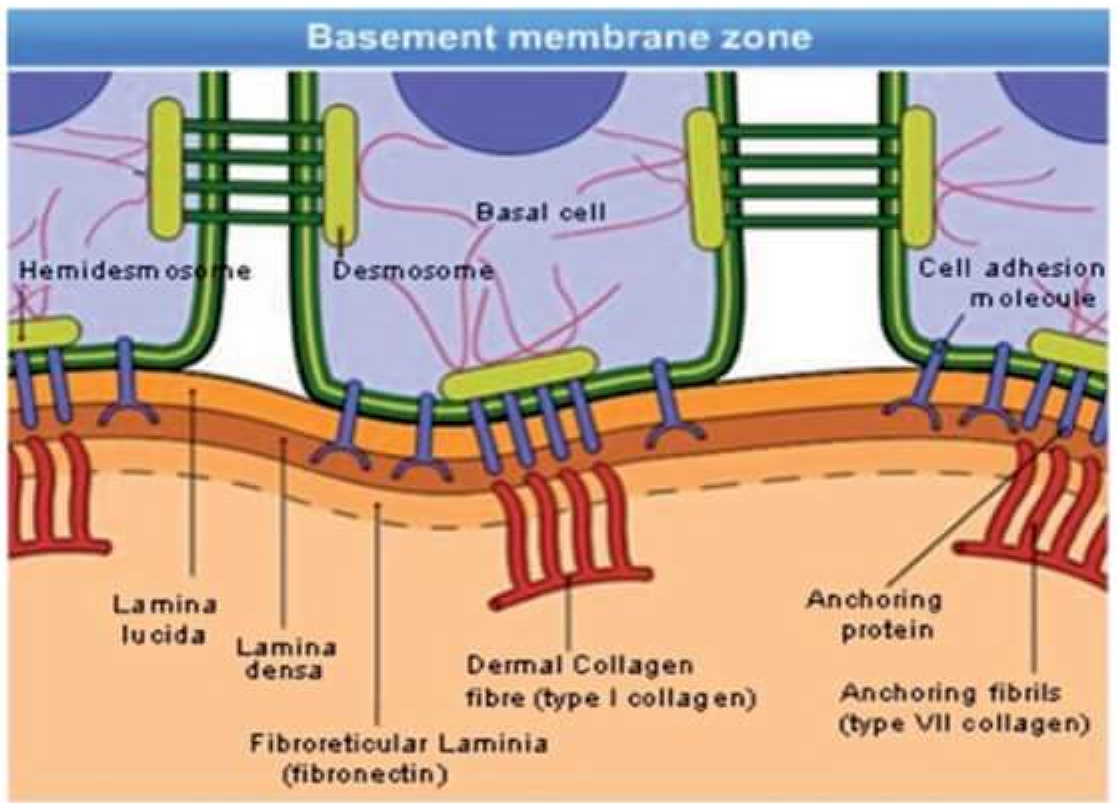
- It is present between epidermis & dermis layer

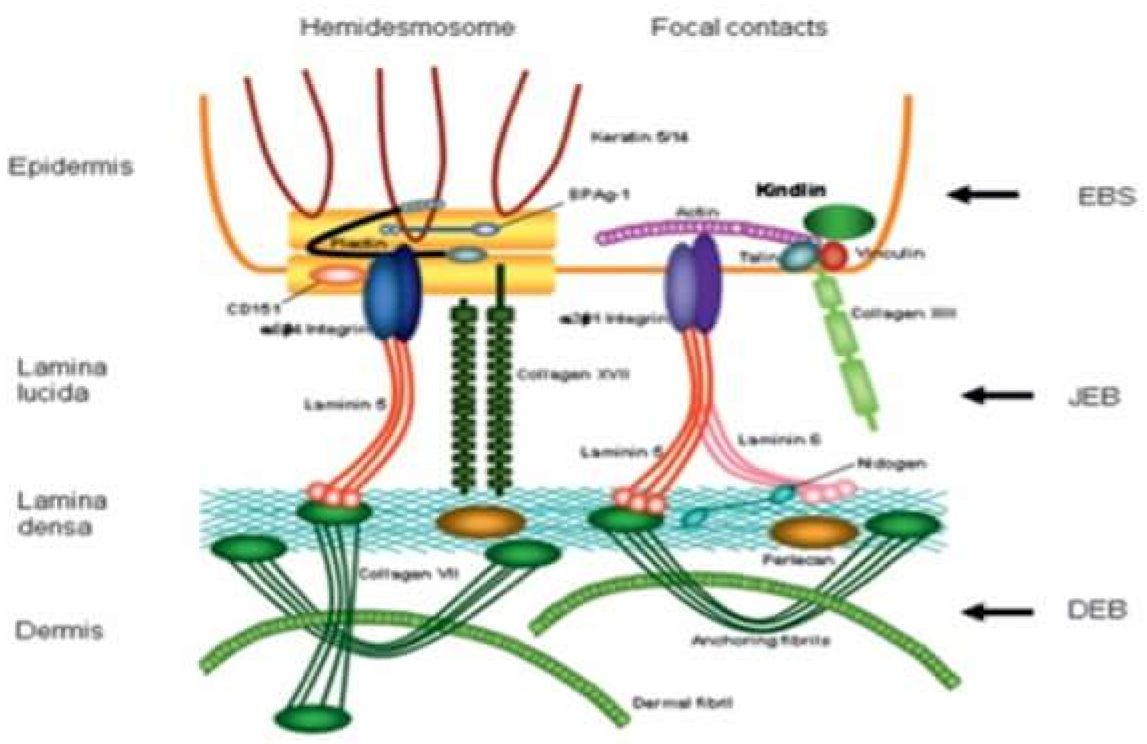


- *Weakest layer is Lamina lucida / salt spilt layer.*
- Defect in these proteins leads to subepidermal disorders.

Subepidermal Disorder:-

Proteins	Defect
1) BP Ag – 1 Or BP 230 (Molecular weight 230 kilodalton)	- Bullous Pemphigoid – most common – BP Ag 2 > BP Ag – 1 - Dermatitis Herpetiformis
2) BP Ag – 2 Or BP- 180 / Collagen 17 (Molecular weight – 180 kilodalton)	- Bullous pemphigoid - Liner IgA disease - Lichen planus pemphigoides - Herpes gestationalis
3) Collagen – 7	- Epidermolysis bullosa dystrophicans





DERMIS

Made up of collagen, elastin & reticular fibers in matrix of hyaluronic acid, dermatan & chondroitin sulphate.

→ **Most abundant fibers in dermis are :collagen Type I (70-80%) > Type III (10-15%)**

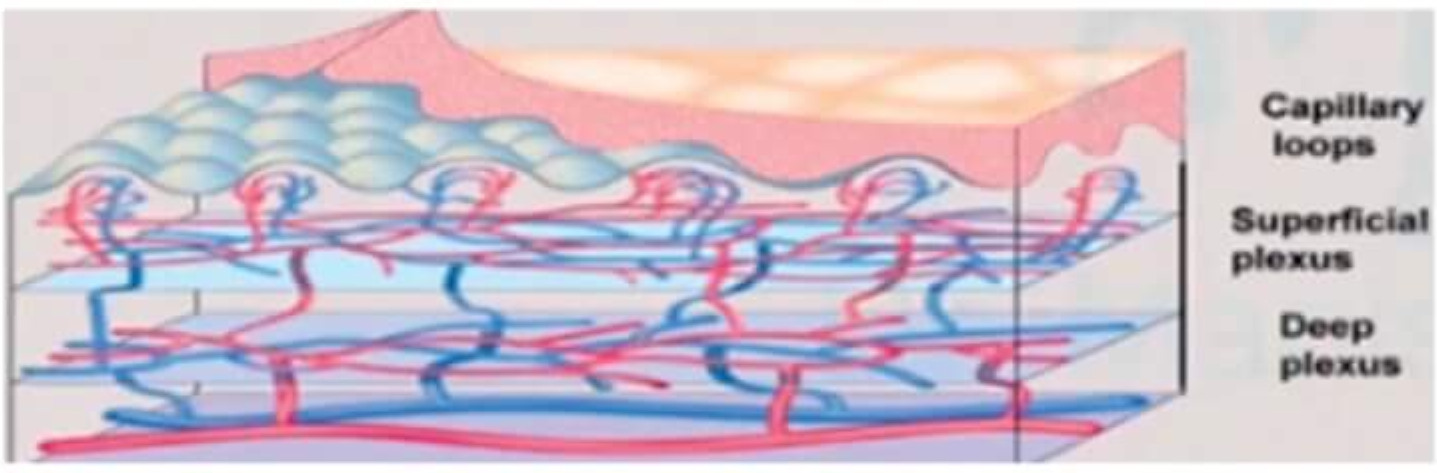
Dermis
→ Papillary dermis → 1/10th portion of dermis
→ Reticular dermis → rest of the dermis

Cells of dermis

→ Most abundant cells present are "Fibroblasts"
→ Other – Macrophages
  - Mast Cells

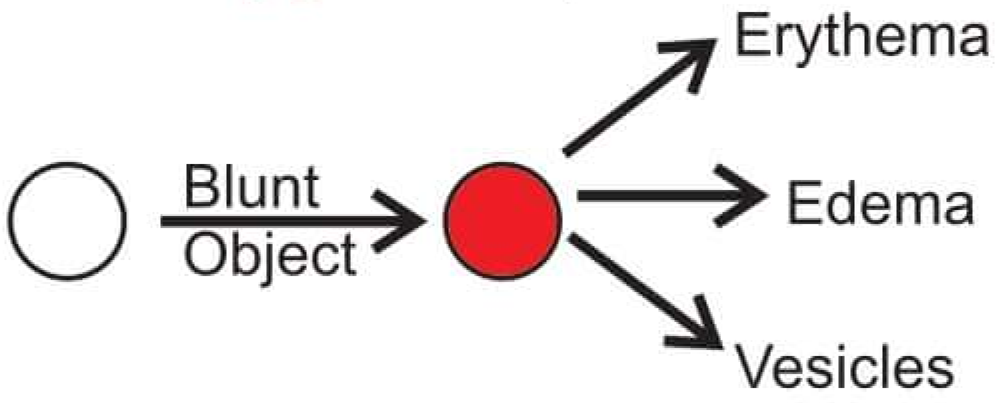
Vascularity of dermis :

→ Capillary loops in dermal papilla
→ vascular plexus { Superficial plexus below papillary layer
  Deep plexus below reticular layer



Important Fact

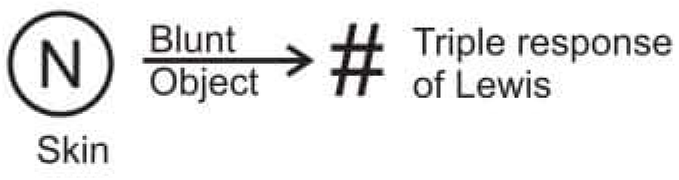
* **Urticaria pigmentosa** (cutaneous macrocytosis) → common in children



* **Darier sign**→ seen in urticaria pigmentosa
(NEET 16/JIP 17/18)

* **Pseudo Darier sign**→ smooth muscle Hematoma (pilo erection)

→ Dermographism is seen in urticaria – Itchy, transient, lines, wheals

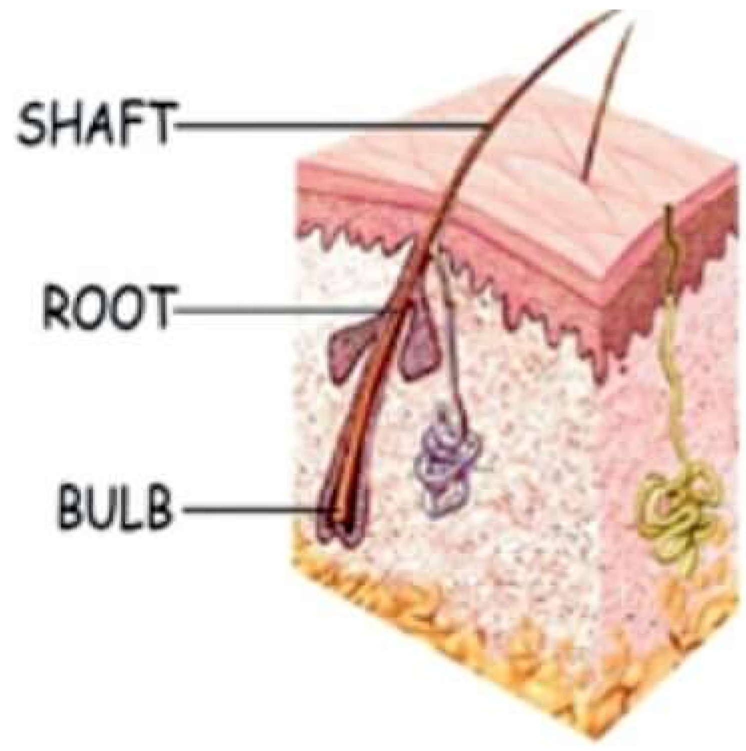


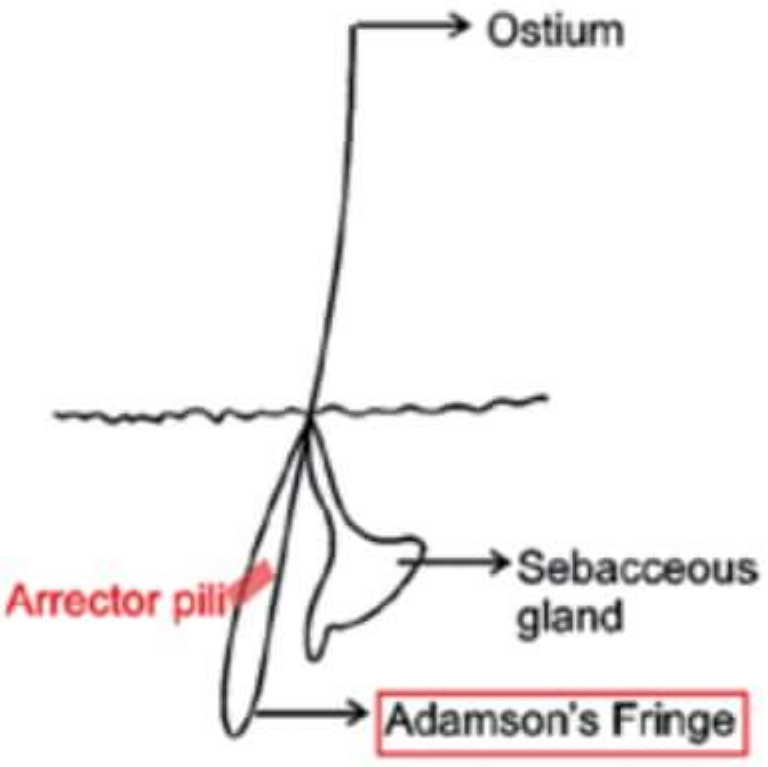
→ White dermographism (vasoconstriction) → Atopic dermatitis
↓
(whenever skin is rubbed white line is seen)

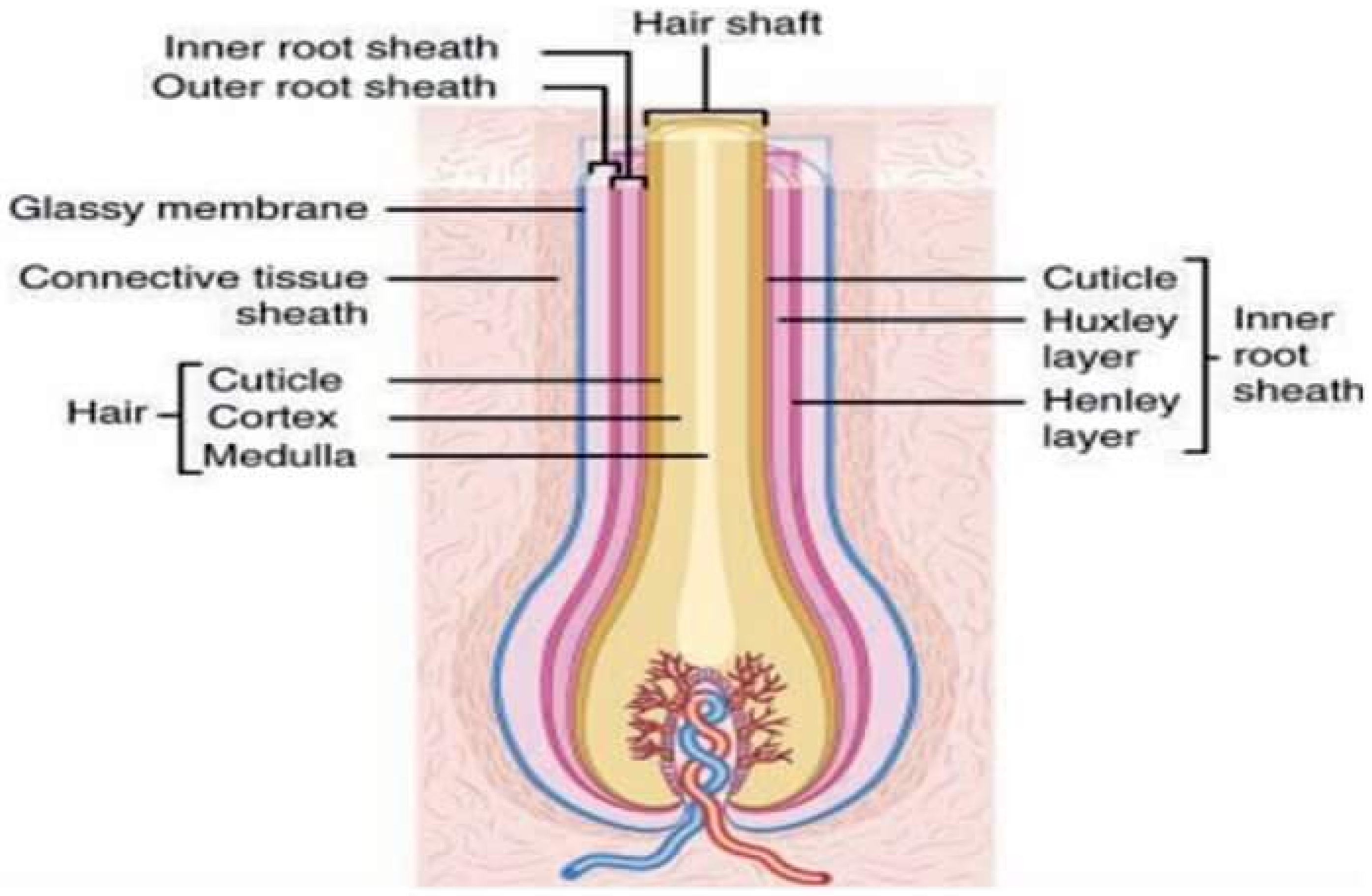
STRUCTURE OF HAIR AND HAIR DISORDERS

Appendage of skin

- → Hairs
- → Nail
- → Sweet gland
- → Sebaceous gland







Hair follicle Structure :

UPPER HAIR FOLLICLE
- I. INFUNDIBULUM → 1. OSTIUM ↕ 2. SEBACEOUS DUCT OPENING
- II. ISTHMUS → 2. SEBACEOUS DUCT OPENING ↕ 3. ARRECTOR PILI ATTACHMENT

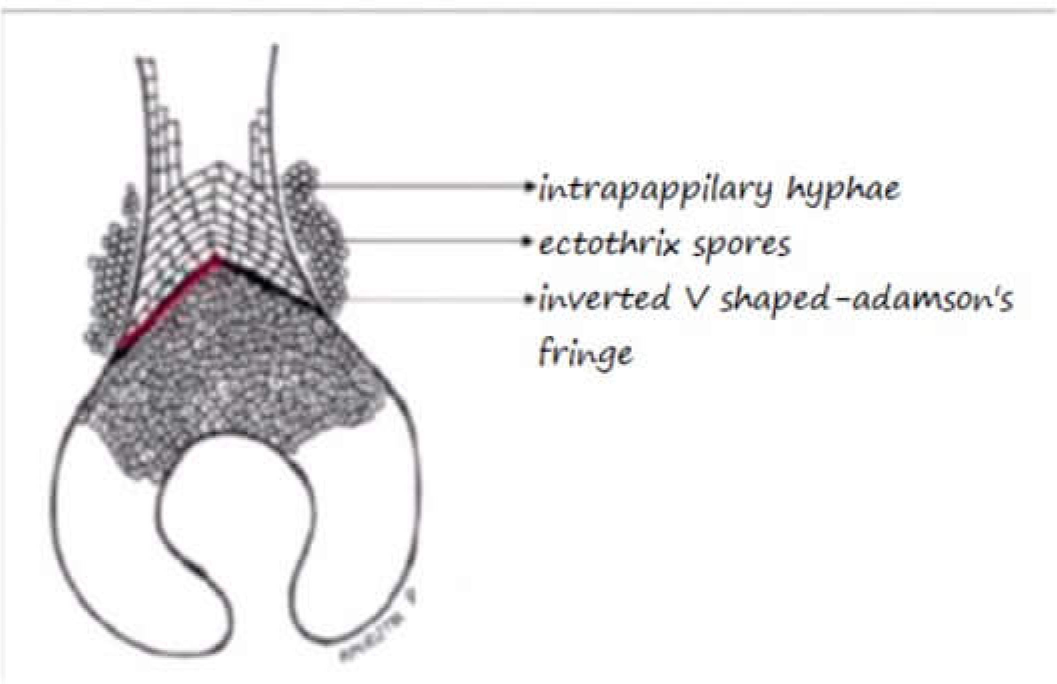
LOWER HAIR FOLLICLE
- III. STEM → 3. ARRECTOR PILI ATTACHMENT ↕ 4. ADAMSON'S FRINGE
- IV. BULB → 4. ADAMSON'S FRINGE ↕ 5. BASE

→ Conclusion based on attachment of arrector pilli muscle hair follicle is divided into upper and lower hair follicle.

Q. DNB Adamson fringe is a component of

A: bulb

Stem cells are also part of bulb and
it is inverted V shape

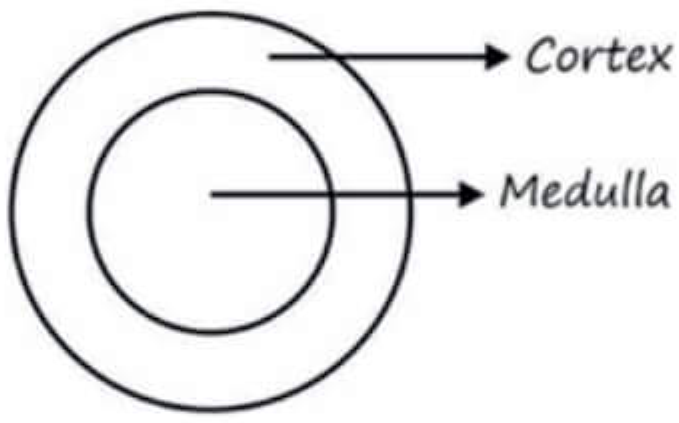


→ Adamson noticed, while studying cases of tinea capitis that fungal hyphae and spores never intruded beyond adamson's fringe as it's a highly keratogenous part, and the stem cell (prolipotent cells) are also present here in this highly keratogenous part of upper portion of hair bulb with inverted V shape

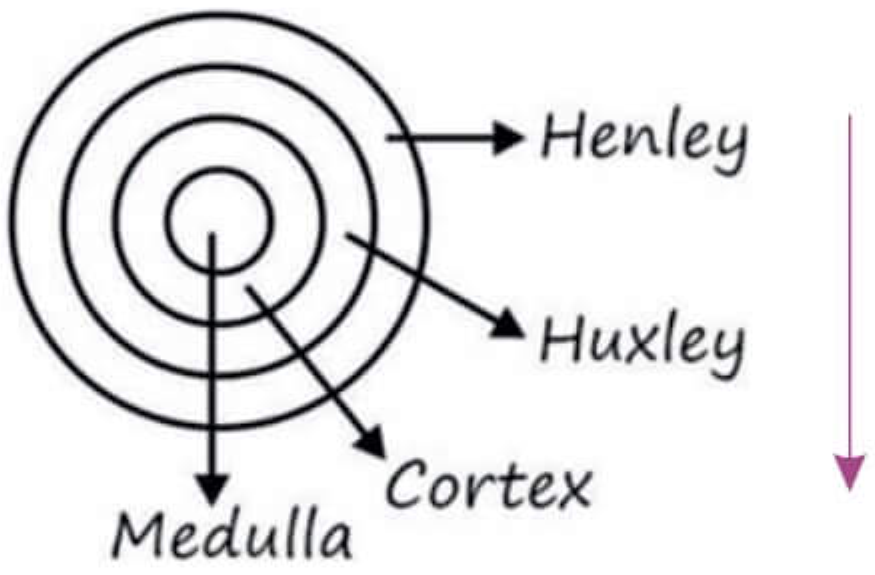
Q. Medullary index in human hair?

Ans: 1: 3 Humans [1:2 Animal]

→ Medullary Index = Ratio of medulla: Cortex of hair



Note: Henley, Huxley, cuticle are layers of hair follicle



Types of Hair follicle:

1. Lanugo- seen in fetus
2. Vellus
3. Terminal

Vellus	Terminal
- Medulla absent	- Medulla present (thick)
- Melanin absent	- Melanin present (dark)
- Depth - till Dermis	- Depth - subcutis (strong)
- Seen in pre pubertal age groups	- Seen in adults

Hair cycle - 3 Phases→ **(Mnemonic-ACT)**

Anagen ⟶ **Catagen** ⟶ **Telogen**

Anagen	Catagen	Telogen
- Growth phase	- involution phase	- Resting phase
- 3 years	- 3 weeks	- 3 months
- 85% hair	- 1% hair	- 15% hair

A C T →

3 Y W M ←

Hair disorders

1) **Anagen effluvium:** - Diffuse shedding of anagen hair seen after chemotherapy

↓

Shedding

2) **Telogen effluvium:** - Increased telogen hair loss seen in 3 months after pregnancy, typhoid fever or any chronic illness.

3) **Androgenic alopecia**- DHEA

↓ 3β hydroxy dehydrogenase

Androstenedione

↓ 17β hydroxy dehydrogenase

Testosterone

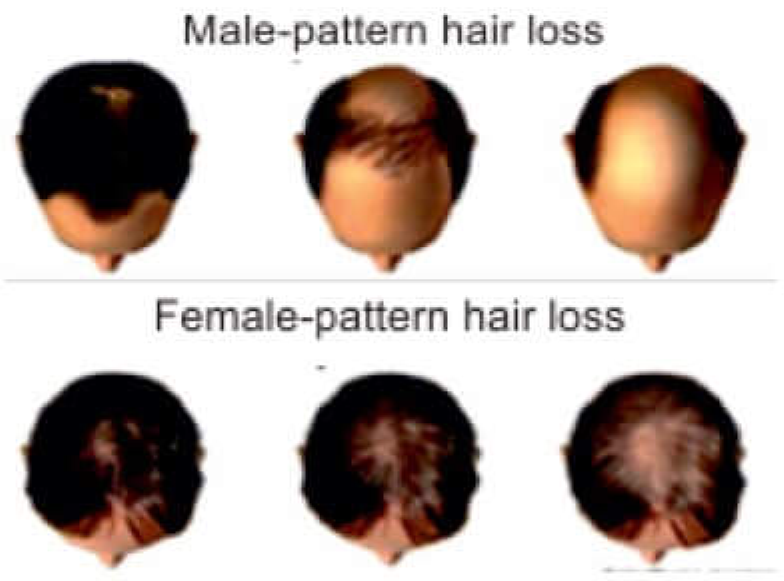
↓ 5α Reductase

DHT → **most potent form- responsible for hair loss**

C/F: - "Patterned Hair Loss"

Male pattern

1. Hamilton Pattern
   - • frontal hairline recedes backwards +
   - • temporal recession +
   - • hair loss from vertex
- Miniature hair seen in reduced anagen phase
- vellus hair increases & Terminal hair decreases In sites involved.



Female Pattern

1. Ludwig Pattern
   - -frontal hair line maintained but hair loss from frontal & vertex portion.
2. Olsen pattern
   - - frontal hair line maintained but hair loss from partition site.

Treatment:

1. Topical: - Minoxidil

MOA 1) increased vascularity (vasodilator, k+ channel opener)
2) Increased anagen phase

(US-FDA approved agent) used in - Male 5%
- Female 2%

2.Systemic: -

I. 5α Reductase inhibitors
E.g:- Finasteride 1mg OD
(used in males, not in females)
It inhibits type II 5α Reductase

(dutasteride is both 5α Reductase type I&II used in Rx BPH)

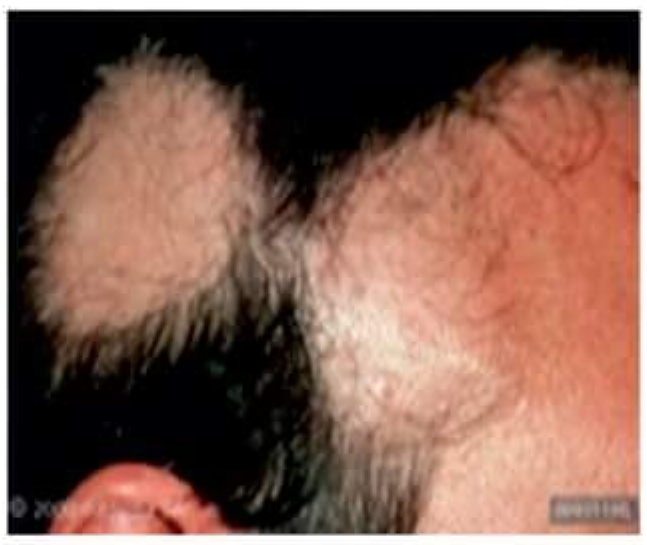
ii. Androgen receptor blockers: e.g. Flutamide
- side effects- hepatotoxicity
- can be used in males and females
- 2nd and 3rd line of therapy

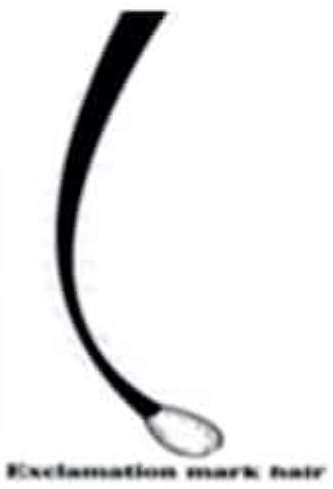
iii. Anti- androgen (side effect in males)
therefore, used only for females
a) cyproterone acetate
b) spironolactone

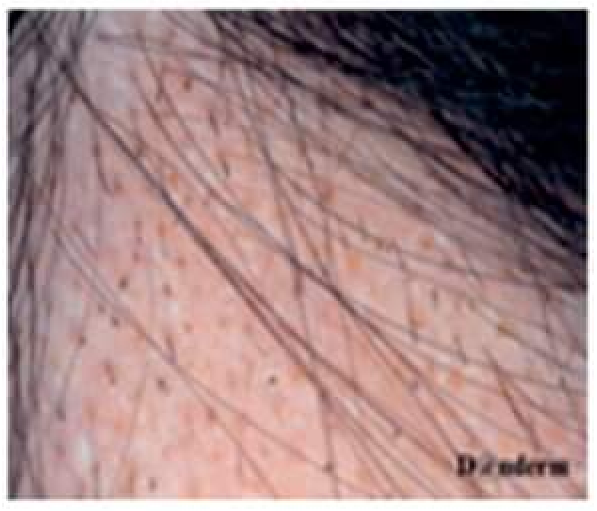
Surgical treatment: hair transplants
a) follicular unit extraction (commonly used, advanced no surgical scar)
b) follicular unit transplant (surgical scar left)

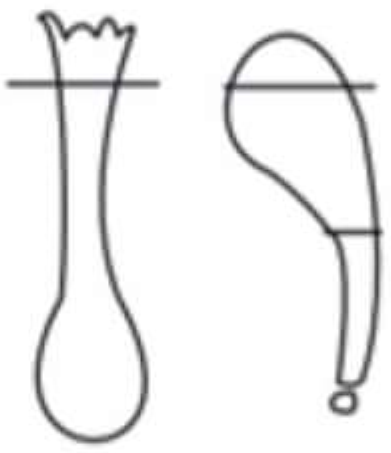
4) Alopecia areata

- It is an autoimmune condition
- C/F: - Patchy hair loss
- On microscopy surrounding these patches show **Exclamation mark sign-** These are broken hair follicle With broad distal & thin proximal shaft.
- It is pathognomonic but not always found.



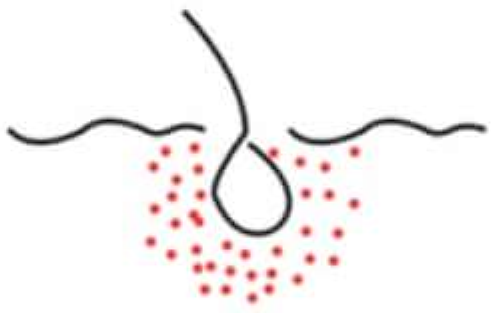






Turning grey phenomenon: -
- It is sparing of grey hair & destruction of melanocytes

Histologically: Perifollicular/peribulbar lymphocytic infiltrate giving swarm of bee appearance.



Treatment: -

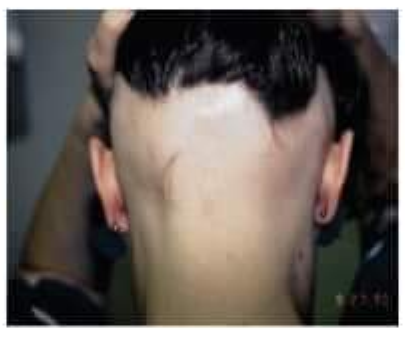
A) Topical: 1. Steroids
  2. Calcineurin inhibitors like-Tacrolimus
  3. Contact irritants/sensitizers: a) Dinitrochlorobenzene
  ↓ b) squaric acid dibutyl ether

They are potent allergens, since Ab forming against the hair follicle and on application of these potent allergen all these Ab attack these allergen. Allowing the hair follicle to grow.

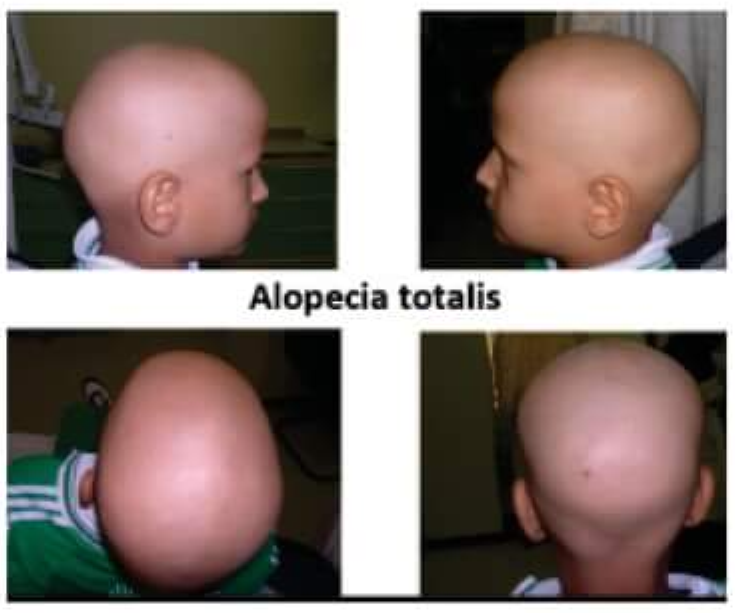
B) Systemic
  a. Steroids: - Oral mini pulse given 2 days/week (Betamethasone) to avoid side-effects.
  b. Azathioprine: - Steroid sparing agent
  c. Levamisole: - immunomodulator

C) intralesional: preferred for single lesion/ on patch triamcinolone long acting steroid-10mg/ml injection at site

D) phototherapy for generalised cases like
  whole Body involvement= A. Universalis
  whole Scalp involvement= Alopecia Totalis

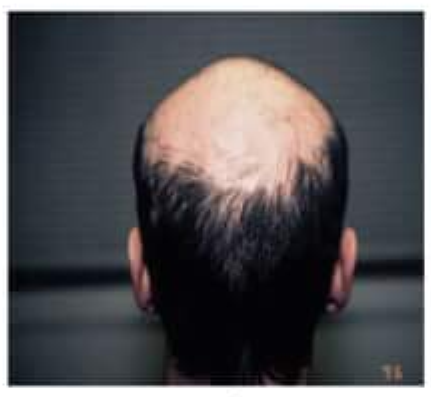


Ophiasis
(Periphery involved first)



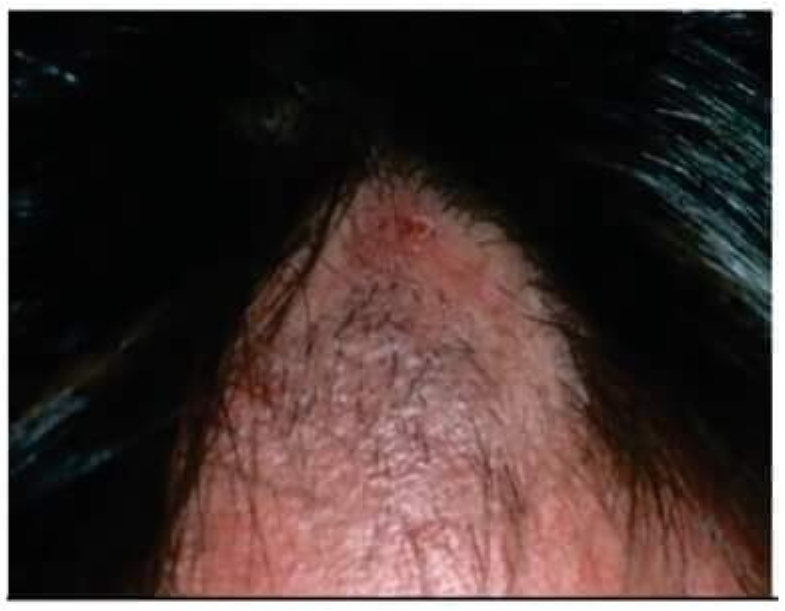
Alopecia totalis

sisaipho
(central portion involved first)



Important Facts: -

1. **Trichotillomania: -**
   - it's a psychiatric condition usually seen in girls, may also be seen in boys and adults
   - **Patient presents with** Patch with variable length of broken hair follicles usually localised
   - **It is an impulse control disorder, patients may eat these pulled hairs and gradually suffer with intestinal obstruction" trichobezoars"**
   - **Diagnosis:** (done in the past) with skin window test: a small patch of hair is shaved off and patient called after 2weeks, on examination its found that all the hair follicles are normal and of equal length(as hair is short so patient cant pull it) and no defect or disorder of hair is seen so it confirms the diagnosis.
   - on histology: 1) No inflammation seen unlike alopecia areata
     2) perifollicular haematoma/ haemorrhage
     3) trichomalacia: is soft incomplete keratinised hair shaft
   - **Treatment** – TOC - behavioural therapy



2. loose anagen syndrome:

→ It's a autosomal dominant condition

→ Benign self-limiting condition where anagen hairs are easily and painlessly extracted.

→ Usually reported in childhood

→ The presence of anagen hair devoid of its sheath and with 'floppy sock appearance' is a characteristic feature.

→ Loose anagen hair (LAH) can be seen in normal population and in alopecia areata.

→ Confirmation of diagnosis: if the percentage of LAH in LAS is more that 50%.

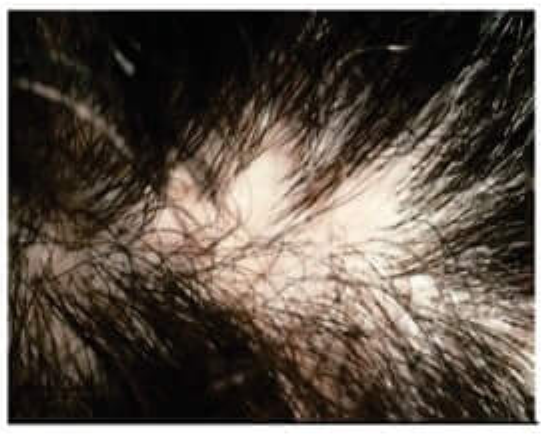
3. Pseudopelade of Brocq:

→ It is an example of scarring alopecia

→ Destruction of the hair follicle produces multiple, round, oval, or, irregularly shaped hairless, cicatricial patches of varying size gives rise to "footprint in the snow appearance".

→ Inflammation is completely absent

→ No pustules, crusts or broken hair follicles



4. Alopecia

Alopecia

- Scarring
- Non-scarring
  - Primary (Mnemonic 'AT')
    1. Anagen Effluvium
    2. Telogen Effluvium.
    3. Alopecia Areata
    4. Androgenic alopecia
    5. Trichotillomania
    6. Tractional alopecia/Hair style alopecia
    7. Tinea capitis(non-inflammatory)
  - Secondary
    1. SLE
    2. 2° Syphilis(Moth eaten alopecia)
    3. Hypothyroidism
    4. Hyperthyroidism
    5. Hypopituitarism
    6. Deficiency of iron, zinc, biotin

5. Acrodermatitis enteropathica:

→ Condition where dermatitis of acral part ie hand, feet, face, groin

→ Condition arises due to deficiency of Zinc.

→ Associated with diarrhoea.

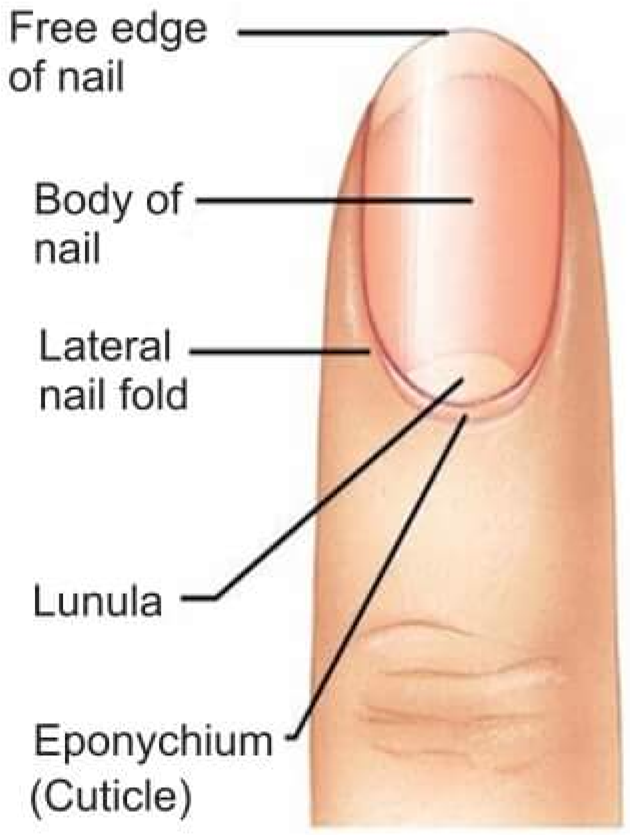
→ Treatment- zinc supplementation

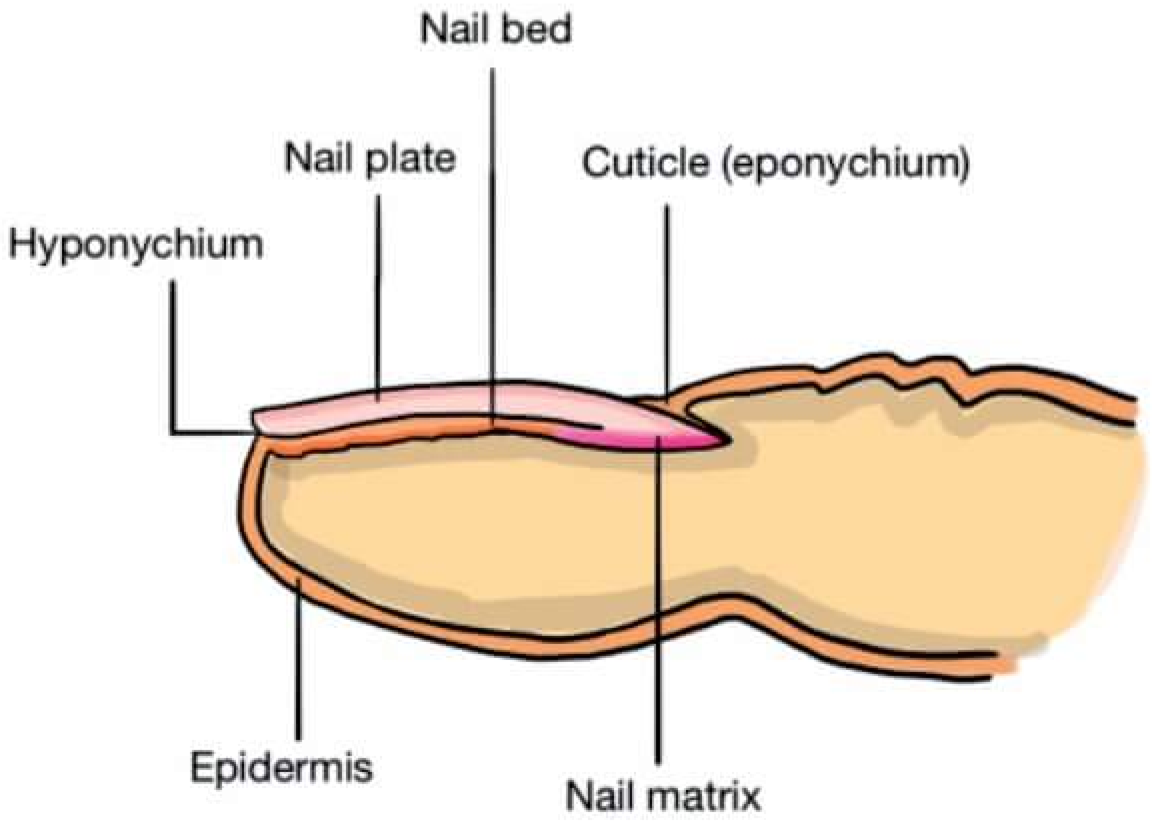
6. lupus erythematosus:

Systemic lupus erythematosus (SLE)	Discoid lupus erythematosus (DLE)
→ It causes non scaring hair loss → But **Late in SLE** scarring alopecia occurs.	→ it causes scarring alopecia always ^{Q} → associated with, - Carpet tack sign ^{Q} - Scaly ^{Q} lesions with hypopigmented centre and hyperpigmented margins - Aggravates on sun exposure ^{Q} - Treatment by hydroxychloroquine's ^{Q}

STRUCTURE OF NAILS AND NAIL DISORDERS

NAILS

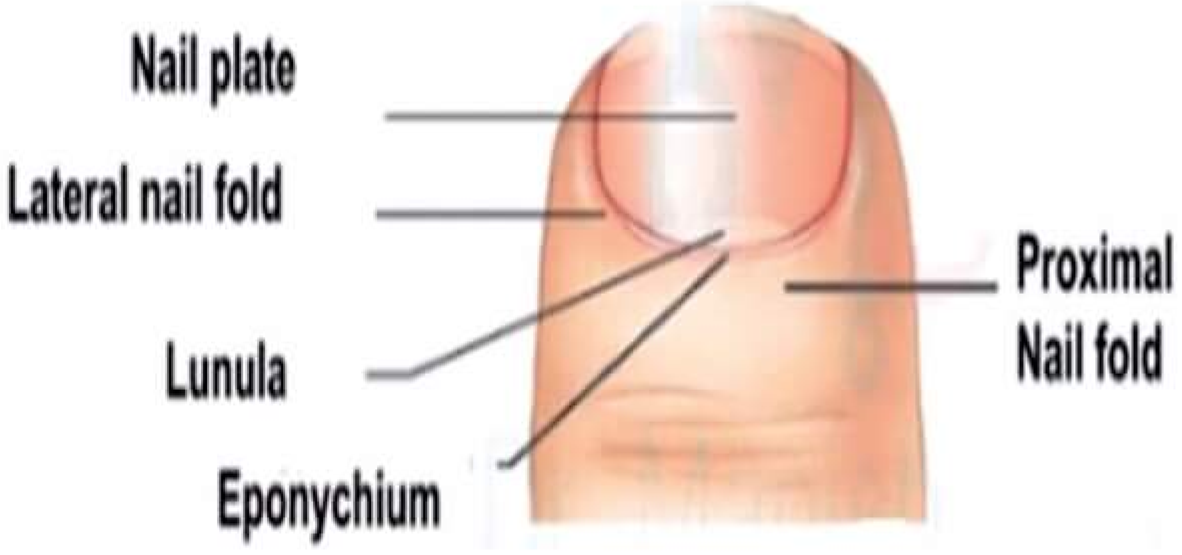




- Nail Plate→ Nail develops from Matrix
- Lunula→ Visible Nail Matrix
- Paronychia-> infection of Nail fold [acute- due to staph aureus, chronic- due to candida infection]
- Hyponychium→ (M/C) site for onychomycosis
- Pitting→ seen due to defect in Proximal nail matrix

Pitting seen in
i) Psoriasis- Thimble pitting (irregular deep pits)
ii) Alopecia areata (regular superficial pits)
iii) Eczema

Structure of Nails:



Lunula - **proximal nail plate with matrix**

Eponychium- **stratum corneum over nail plate**

Hyponychium-**skin below free edge of nail plate**

Nail matrix- **produces nail plate**

Nail bed- **skin without appendages and hypodermis**

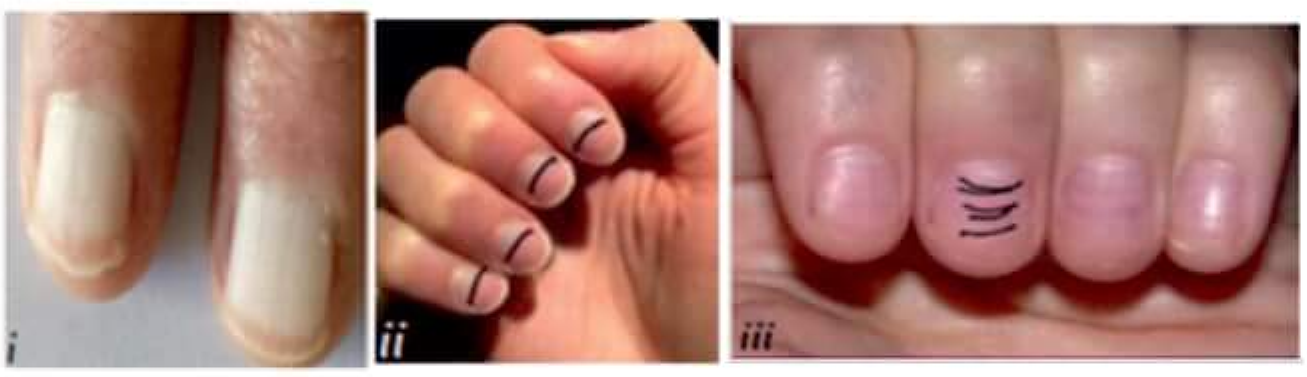
Nail changes in psoriasis: -

1. M/C – Pitting
2. M/Ch-Oil drop sign/Salmon patch- (due to defect in nail bed)
3. Onycholysis (separation of the nail plate from the nail bed)
4. Leukonychia (True)
5. Sub ungual hyperkeratosis (thickening of nail bed)
6. Nail tunnel → (M/C in Onychomycosis> psoriasis)

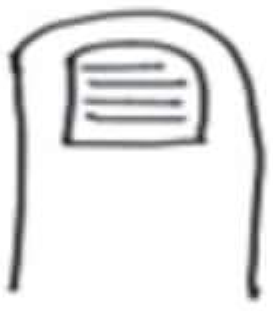
Also seen in onychomycosis

Apparent leukonychia

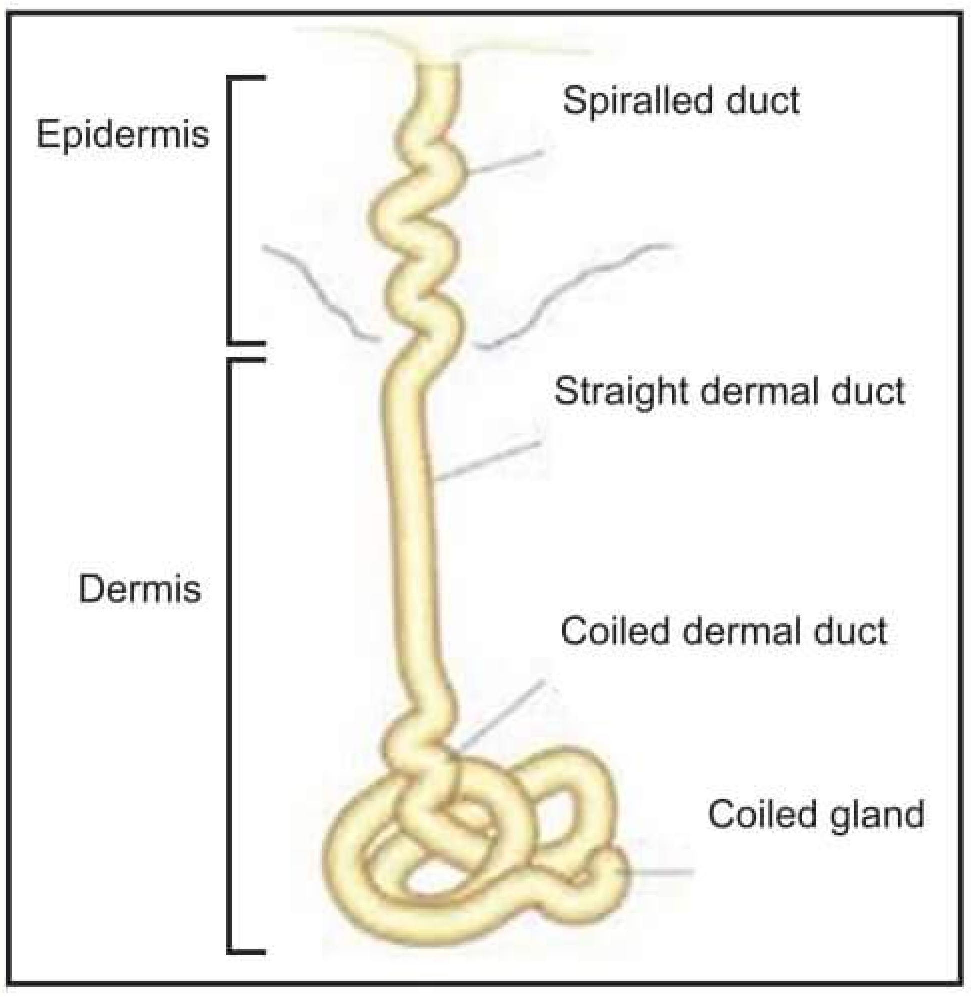
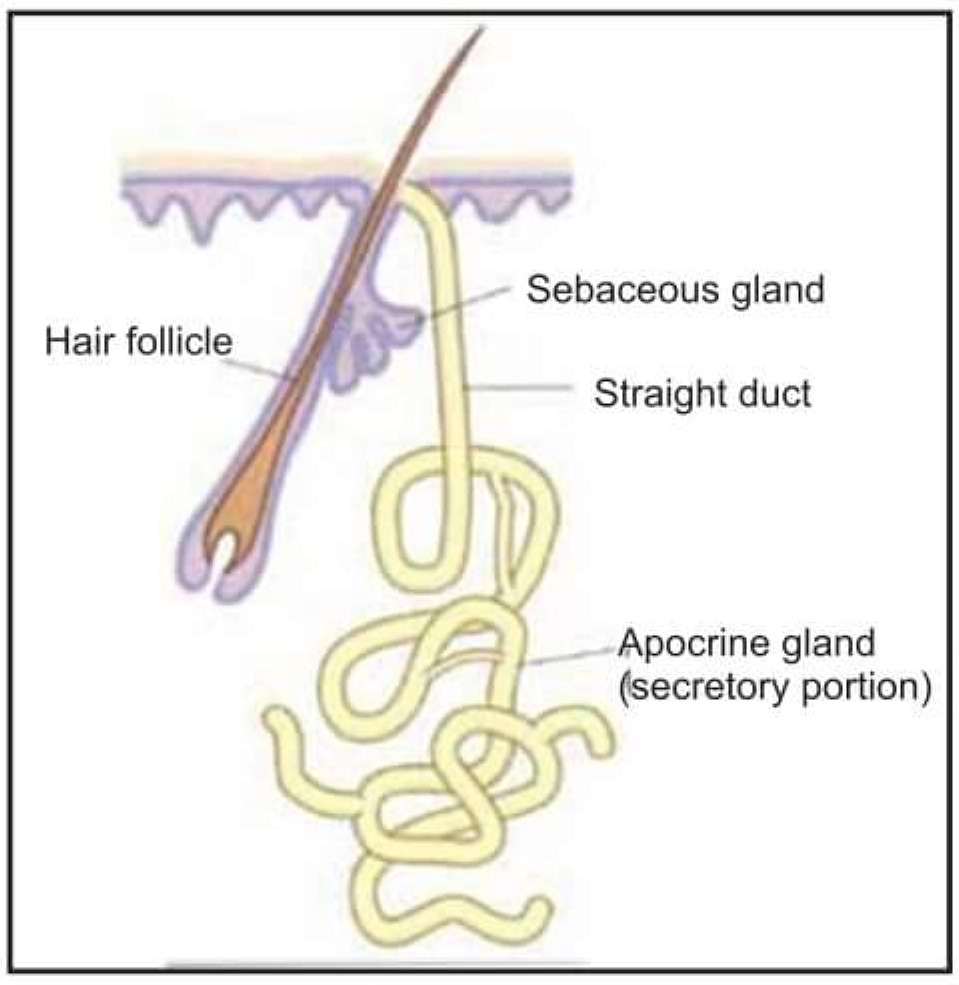
1. Terry nails- Seen in hepatic failure,CHF Q
2. Half and Half nails- seen in CRF Q
3. Banded/Muehrcke nails- Chemotherapy, Hypoalbuminemia



Mees Line- In arsenic poisoning (transverse white lines)



STRUCTURE OF SWEAT GLANDS & RELATED DISORDERS

Eccrine Glands	Apocrine Glands
 Epidermis Spiralled duct Straight dermal duct Dermis Coiled dermal duct Coiled gland	 Sebaceous gland Hair follicle Straight duct Apocrine gland (secretory portion)
1. Duct opens directly On skin	1. Duct open along with hair follicle.
2. Site – Every where	2. Axilla, areola, groin
3. Nerve – Cholinergic Control	3. Adrenergic control
4. Disorders :	
a. Anhidrosis	a. Bromhidrosis (more in apocrine, but also in eccrine)
b. Hyperhidrosis	b. Chlorhydrias
c. Miliaria ← **Both due to blockage in the duct** →	c. Fox Fordyce's disease

Types of Miliria Depending on blockage

Types	Level
1) M. crystalline	Stratum corneum
2) M. Rubra – (most common)	Malpighian layer (stratum spinosum + Stratum Basale)
3) Miliaria profunda	Dermo epidermal junction

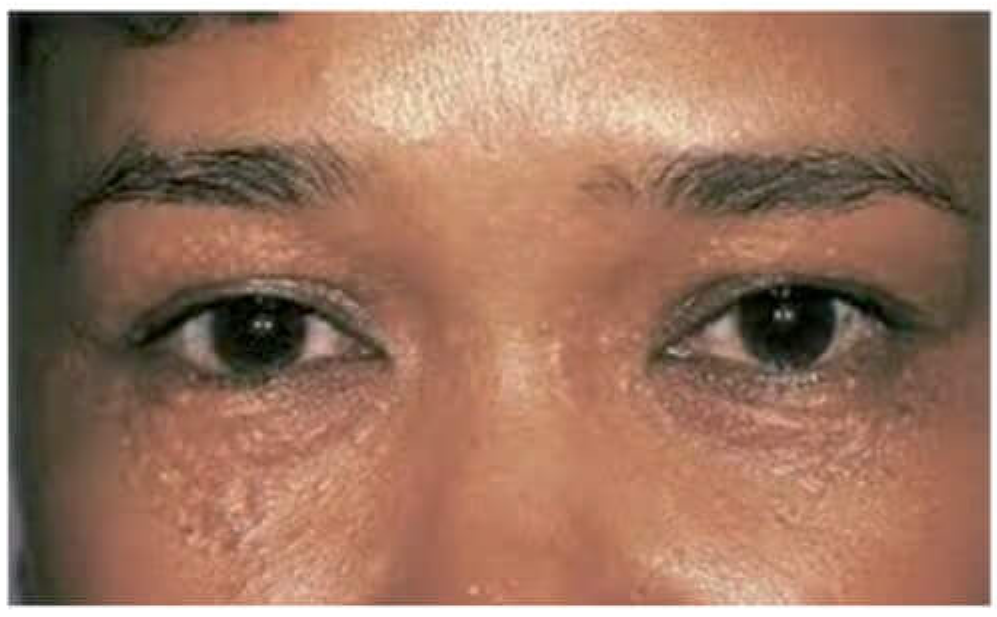
Fox Fordyce disease

- Common in females in 2nd decade
- Itchy papules over axilla

Important fact

Disorders of Eccrine Sweat Gland	Disorders of Apocrine Sweat Gland
1. Hyperhidrosis	1. Bromhidrosis –Foul smelling – most common in Apocrine but can be present in Eccrine
2. Anhidrosis	2. Chromhidrosis
3. **Hidrocystoma**– Malformations leading to retention of sweat	3. Fox-Fordyce's Disease
4. **Neutrophilic eccrine hidradenitis**– Neutrophilic infiltration & degeneration of the eccrine sweat glands. Seen in Acute myeloid leukemia, NonHodgkin's lymphoma undergoing chemotherapy	4. Hidradenitis Suppurativa - seen with multiple sinuses and scarring. - for 1st stage when only nodules & abscess present. - Adalizumab (TNF α inhibitor) [also used in psoriasis]

Syringomas- are harmless eccrine sweat duct tumor (adenomas of intraepidermal eccrine ducts), typically found clustered on eyelids.



SEBACEOUS GLANDS

Present along with Hair follicles
↓ Known as
Pilosebaceous unit
↓
Disorders:

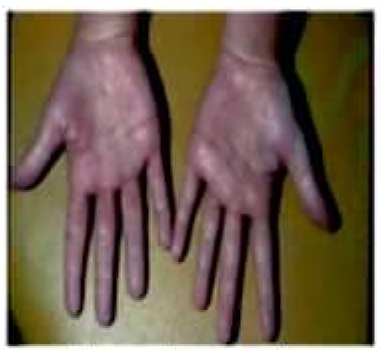
1. Acne vulgaris (M/C)
2. Rosacea

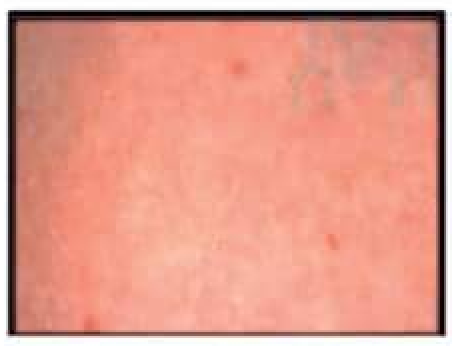
NOTE: Gland of moll (Apocrine sweat gland)- Present in eyelid

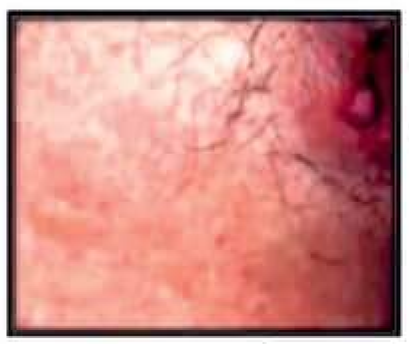
Present alone
↓ Known as
Ectopic sebaceous glands
E.g-on Lips: Fordyce's spots
- Areola: Montgomery tubercles
- Glands: Tyson glands
- Eyelid: Meibomian gland, Gland of zeis

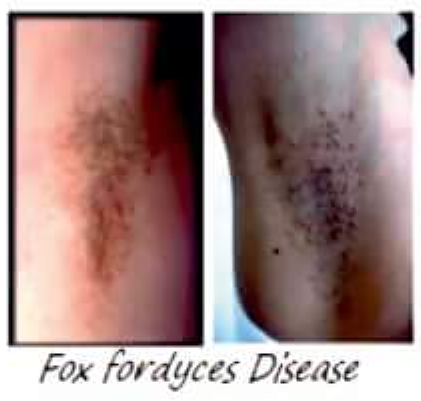
Rosacea

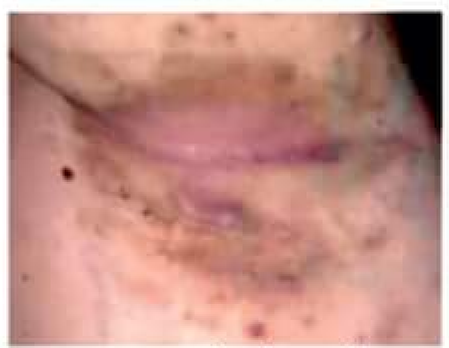
1. Site-convexity of face
2. Type- a) ErythemoTelengeitic (M/C)
    b) Papulo pustular
    c) Phymatous
    d) Ocular
3. Association -Flushing (Aggravate with Heat)

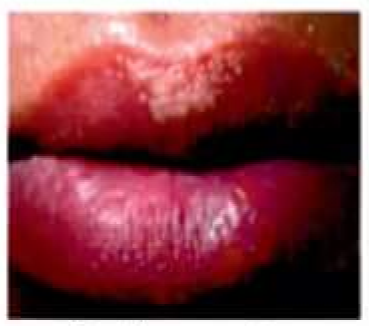

Hyperhydrosis


Miliaria crystallina


Miliaria rubra

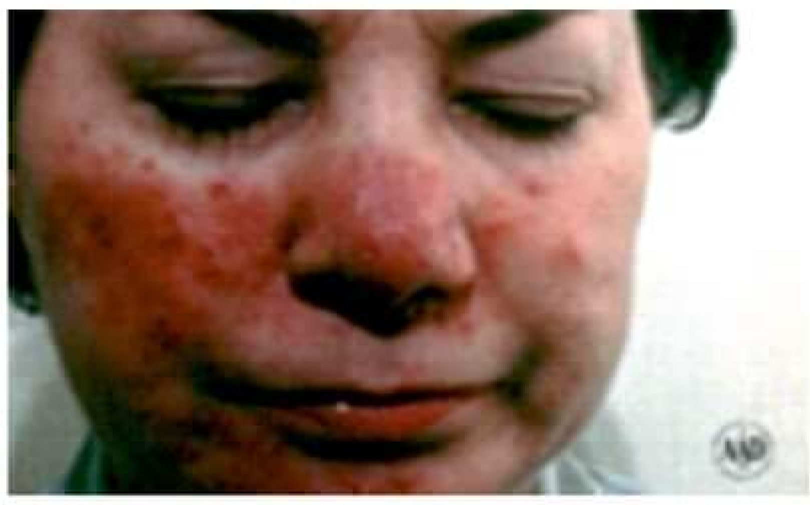

Fox fordyces Disease

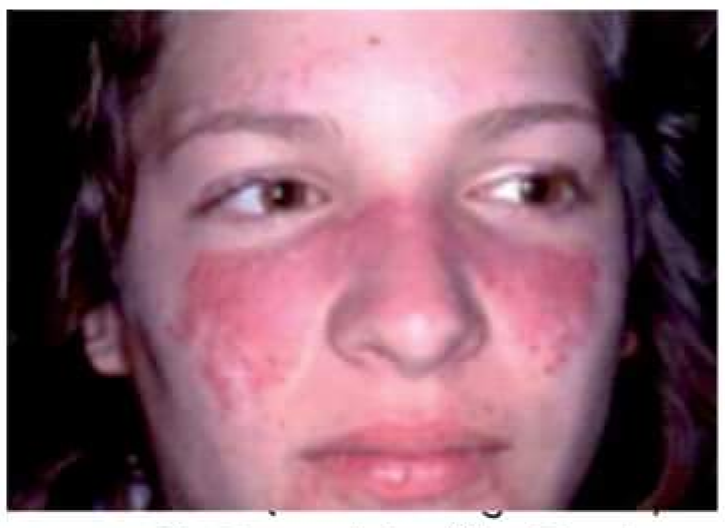

Common site in axilla


Fordyce spots

Differential diagnosis:

- Butterfly Rashes of SLE
- Fixed erythema(on malar eminences and bridge of nose)
- Nasolabial fold spared
- Associated with other systemic manifestations of SLE like-fever, Arthritis


Rosacea


SLE utterfl Rash

ACNE VULGARIS

Etio – pathogenesis:

- Increased Androgens
- Increased IL-1 activity
- Decreased Lenoleic acid

Hyperproliferation of potassium in duct of sebaceous glands

↓

Blockage of duct

↓

Increase sebum collection

↓

Proliferation of Propionibacterium acne

↓

release of Lipase

↓ ↓

Triglycerides | Free fatty acid

↓

Inflammation

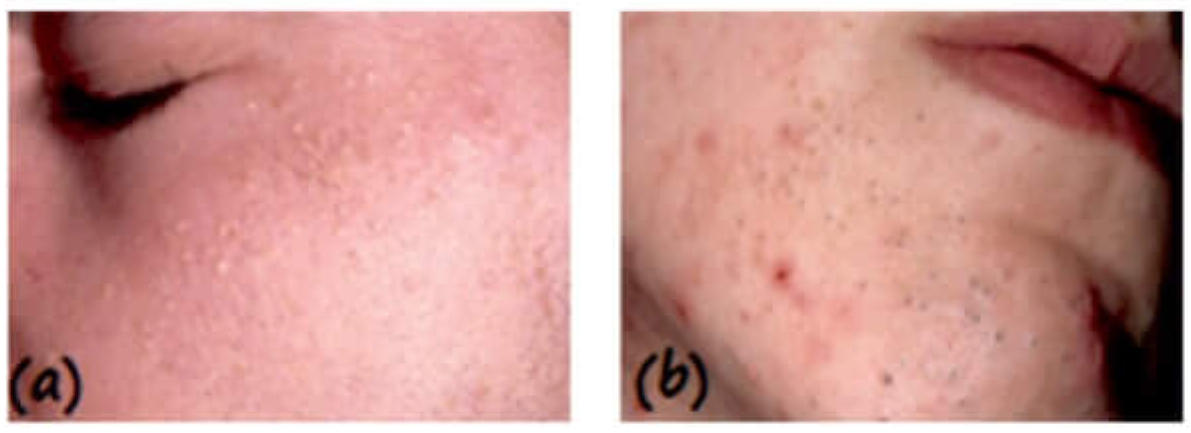
Clinical feature: – **"Polymorphic lesion"**

i. Papules, nodules, cysts, pustules, abscess
ii. Most characteristics- comedones

2 types

↙ ↘

Open/black | Closed/white



Etiology:

Propionibacterium **acne**

↓

Lipase

↓ ↓

Triglycerides | free fatty acids

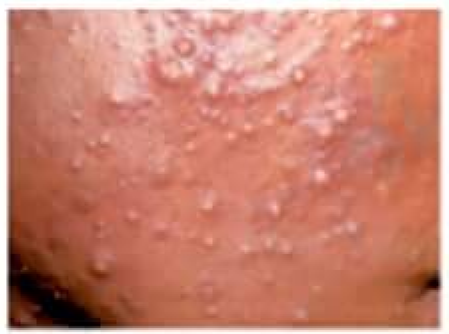
↓

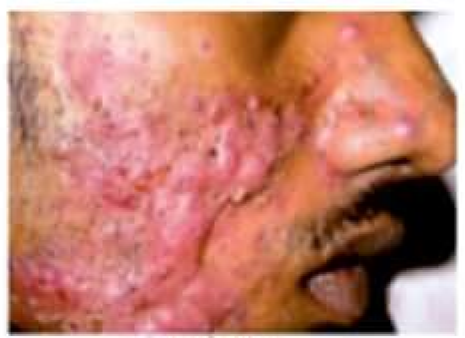
Inflammation

Classification

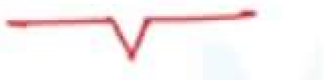
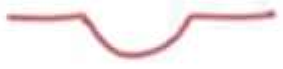
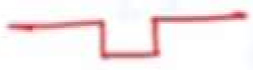
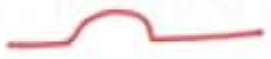
Grade I- Mainly Comedones
Grade II- Mainly papules
Grade III- mainly pustules
Grade IV- Nodules dust abscess
(Most severe also called Nodulo-cystic acne)
↓ DOC
Isotretinoin (Retinoids)
↓
13 cis Retinoic Acid

Dose= 0.5-2mg/kg


Grade II

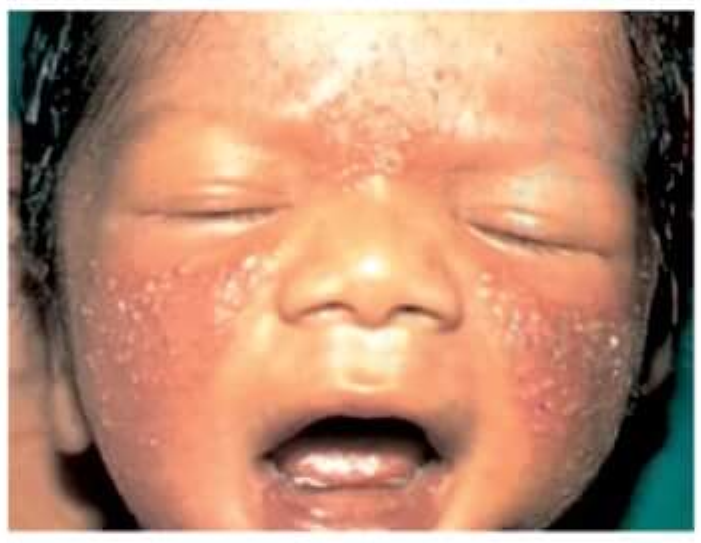

Grade IV

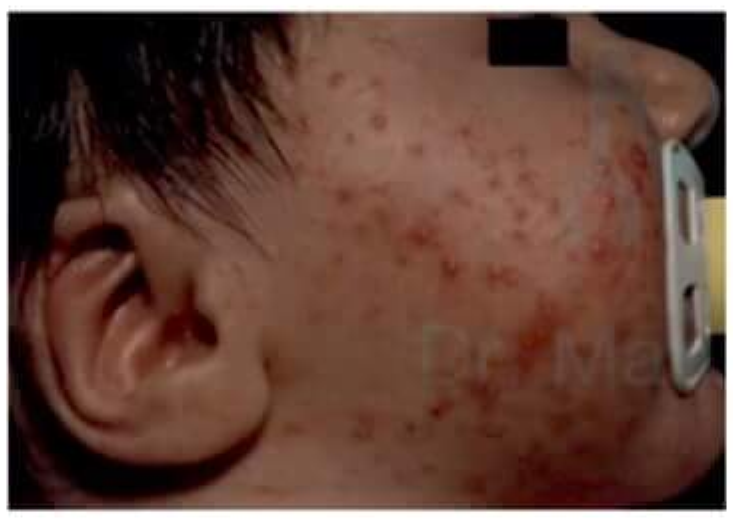
Scarring following acne can be of following types

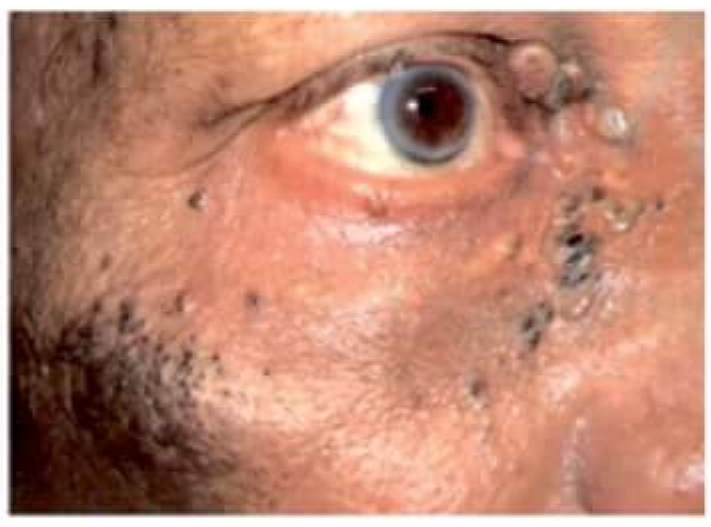
1) Ice pick scars-

2) Rolling scars-

3) Boxcar scars-

4) Hypertropic scars-


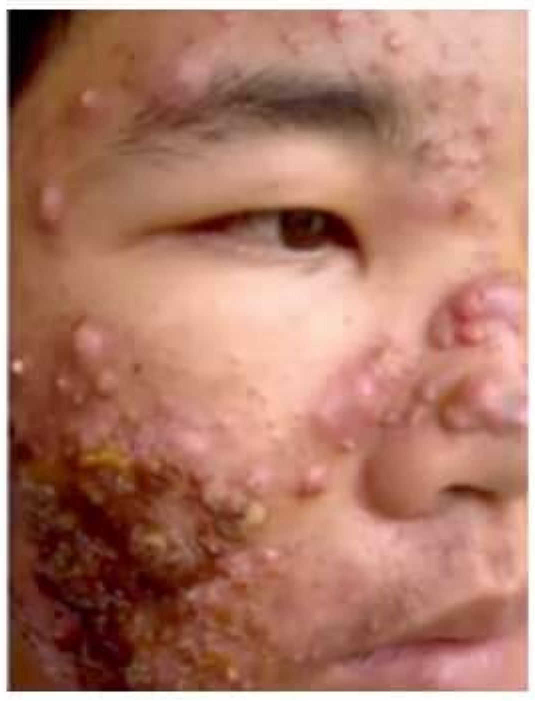
ACNE VARIANTS

1) Neonatal acne – increased maternal androgen
2) Infantile acne- Increased DHEA
3) Senile acne- oldage
4) Acne fulminans- fever
5) Acne excoriee- OCD
6) Morbihan disease -Solid facial edema
7) Acne conglobata- most severe type of acne
8) Drug induced acne- monomorphic acne

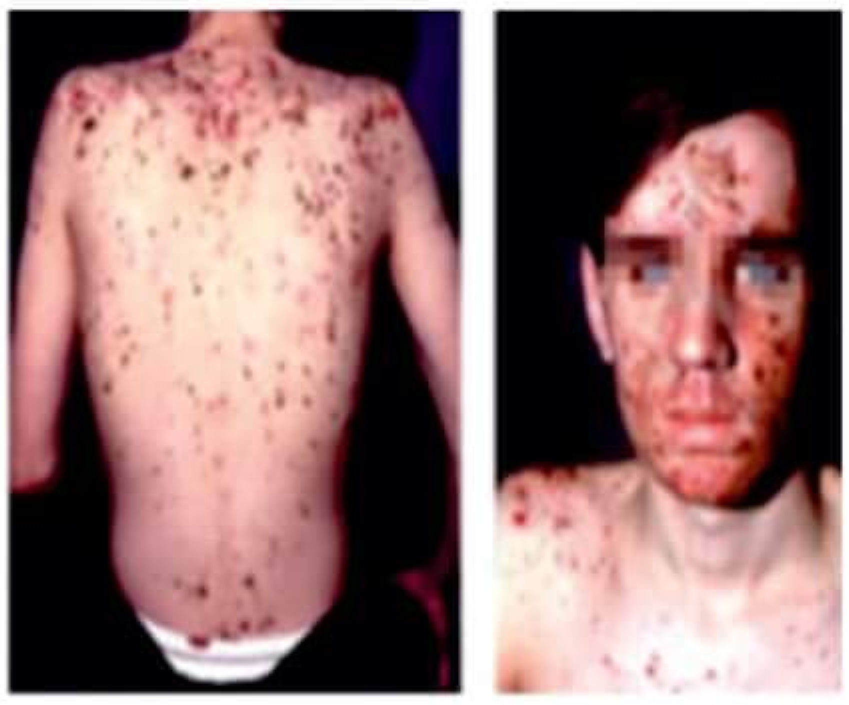

Neonatal acne


Infantile acne

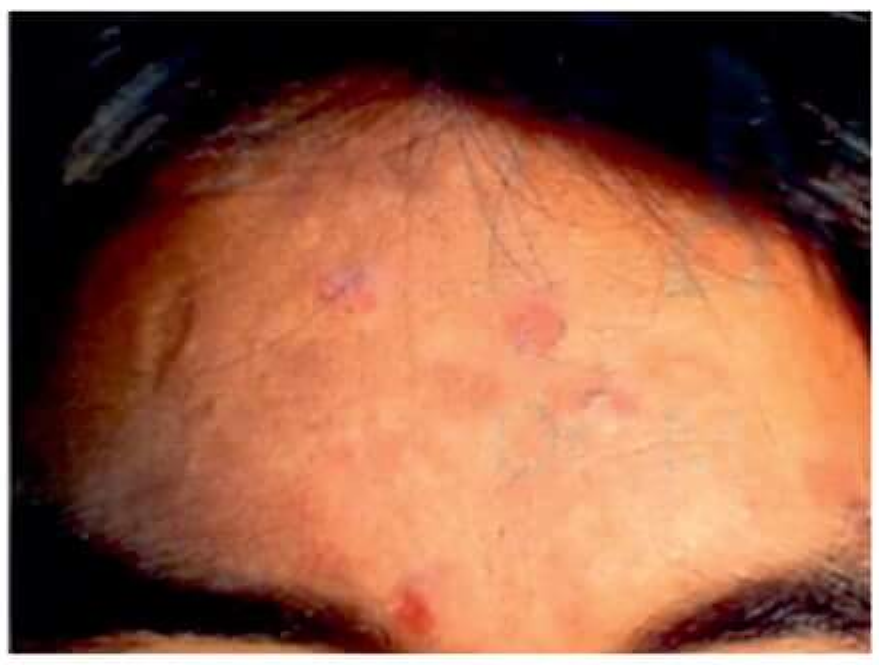

Senile acne



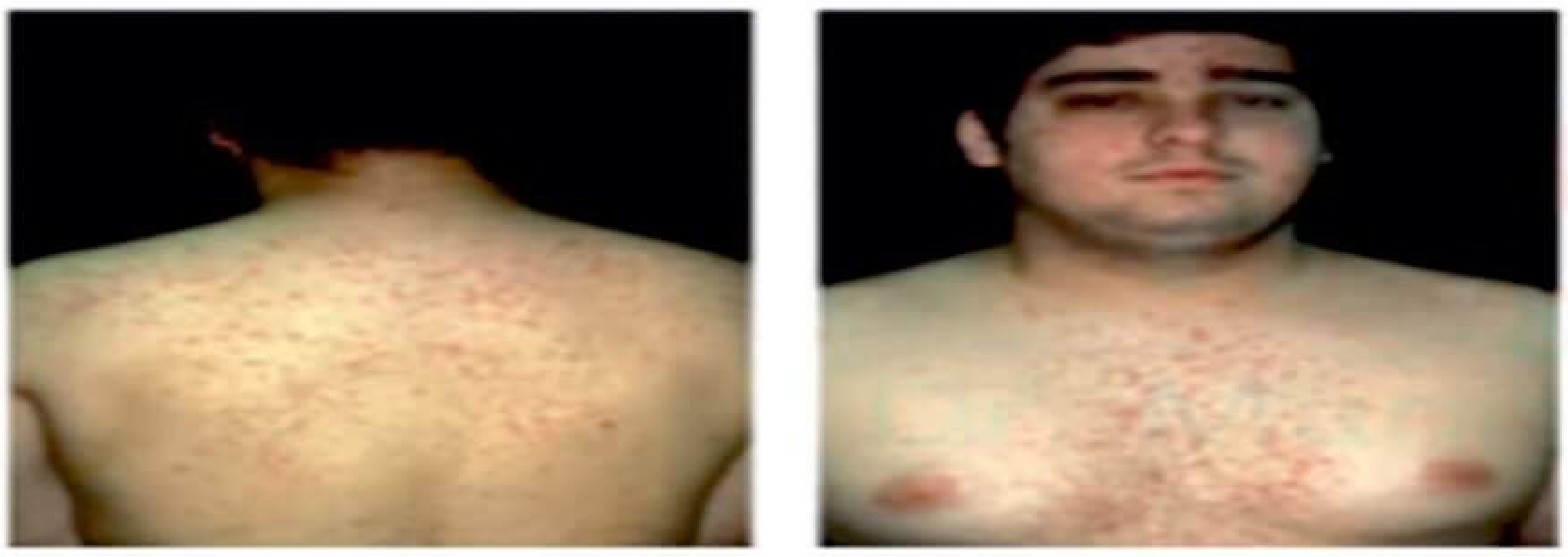
Acne fulminans



Acne conglobata



Acne excoriee



Drug induced Acne

Drug induced Acne

- Steroids (MC in world)
- OCP (MC in west)
- Isoniazid (INH) (MC in India)
- others - Lithium, Iodine,phosphorus

Treatment:

Topical

1. Clindamycin
2. Benzoyl Peroxide
3. Retinoids (adapalene, tretinoin)

Systemic

1. **Antibiotics**
    a. Doxycycline S/E Photo onycholysis
    b. Minocycline S/E Hyper pigmentation of skin & nails Melanonychia (dark nails)
    c. Azithromycin
2. **Retinoids**
    - Isotretinoin-DOC for Severe /Grade 4/Nodulocystic Acne
      Acne conglobata
    - Dose- 0.5 -1mg/kg/Bwt
    - How long can be given- cumulative dose of 120-150 mg/kg/bwt

Side Effect:

1) Dryness of lips (M/C)
2) Teratogenic (It is teratogenic for 1 month after stoppage of drug, so avoided in pregnancy)
3) Hepatotoxic drug

4) Hypertriglyceridemia
5) Pseudotumor Cerebri (Benign Raised intra Cranial Tension)
6) Avoid:Tetracycline vitamin A
7) Avoid in depression (increase suicidal tendency)
8) Most severe side effect- DISH (Diffuse Interstitial Skeletal Hyperosteosis)

Note: "Acitretin"

DOC for pustular psoriasis

Teratogenic effect lasts for 3 years after stopping the therapy

CHEMICAL PEELING

Indications of chemical peeling

I) Melasma
ii) Acne
iii) Acne scar
iv) Photo ageing

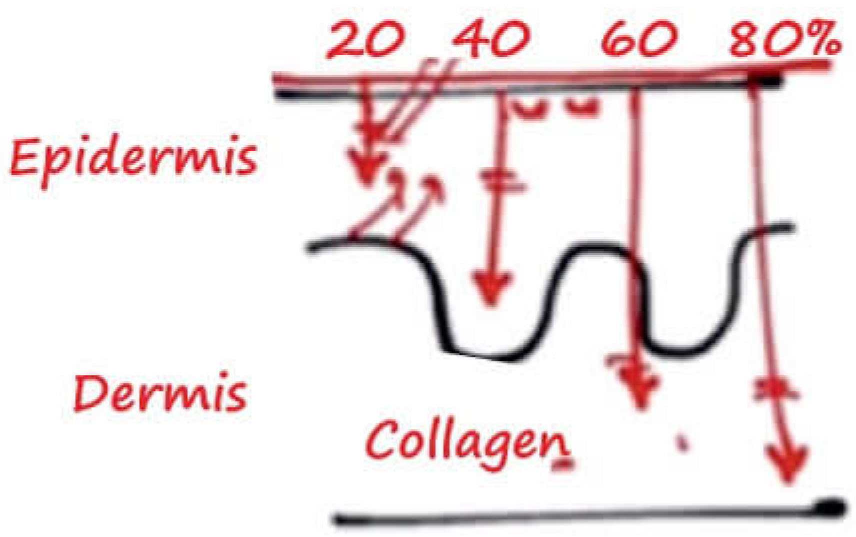
Mechanism of action:

i) Skin resurfacing
ii) Collagen remodeling

→ On acid application to skin it penetrates into epidermis and denature cells
→ Denatured cells then sloughed off.
→ Superficial skin which sloughs off is replaced by normal skin. This is called as skin resurfacing.
→ When the penetration is upto the level of dermis, collagen remodeling starts and acne scars start healing from below.

Agents used for chemical peeling:

1) Pyruvic acid
2) Glycolic acid
3) Malic acid
4) Citric acid } Fruit acids / α OH acid
5) Lactic acid
6) Tarteric acid
7) Salicylic acid / β OH acid
8) Trichloro acetic acid (TCA)
9) Mandelic acid
10) Jessener's solution - combination of salicylic acid + lactic acid+ Resorcinol



BLISTERING DISORDERS

→ Blisters are filled lesions.

If Size
- < 0.5 cm – Vesicles
- > 0.5 cm – Bullae

→ For formation of Blisters there should be a site of separation.

Depending on site of separation

Sub corneal	**Intraepidermal**	**Subepidermal**
Site just below St. Corneum	Site within Epidermis Disease included are e.g. of Acantholysis	Site below Epidermis E.g. diseases affecting DEJ protein

> Any layer just below St. corneum is with in epidermis so sub corneal is also a superficial variant of Intra epidermal

A. Intra Epidermal Blistering Disorders–

1. **Pemphigus**
   - → It is an immune bullous disorder
   - → Immunologically mediated
   - → leading to formation of antibodies against desmoglein causing Intraepidermal bullae.

Type of Pemphigus–

1. Pemphigus vulgaris (MC)
2. Pemphigus foliaceus
3. Pemphigus vegetans (rarest) – variant of pemphigus vulgaris
   MC Site: Axilla, shows–Vegetative lesions
4. Pemphigus Erythematosus– Variant of pemphigus foliaceus
   MC site: Malar eminence erythematous lesion

> In SLE also malar eminences involved and lesions are (red) erythematous (lupus erythematosus)

→ Pemphigus Erythematosus is variant of pemphigus foliaceus PF and has features like lupus erythematous LE, therefore PF+ LE => PE

Others

5. Jogo selvagem: commonly seen in population of Brazil,variant of pemphigus foliaceus
6. Drug induced pemphigus:
   - → MC: Penicillamine (thiol groups in culprit here)
   - → 2nd MC: Captopril
   - → Others: Piroxicam, Rifampicin, Phenylbutazone
   - → Variant of P. foliaceous > P. Vulgaris
7. **Paraneoplastic Pemphigus**

   Seen with
   - → non-Hodgkin lymphoma (NHL)
   - → CLL
   - → Castleman Disease

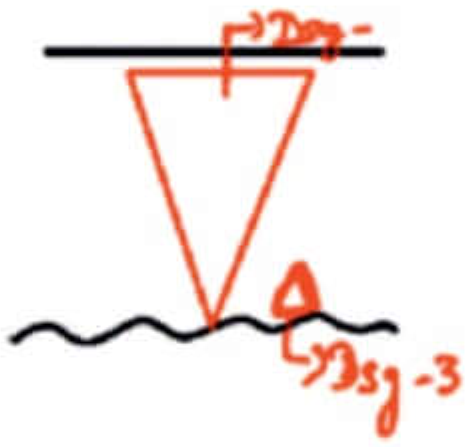
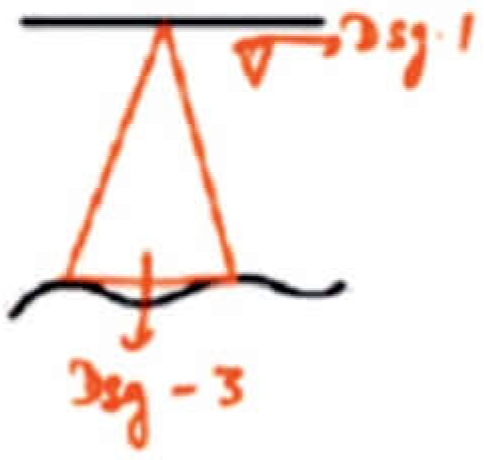
   Others:
   - → Sarcoma
   - → Thymoma
   - → Spindle cell tumour

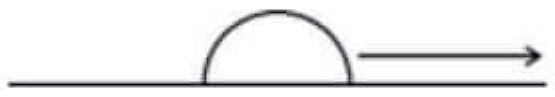
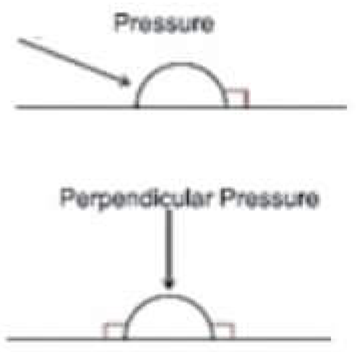
   - → Dx of Paraneoplastic pemphigus is made with the help of Anhalt criteria (mainly for NHL, CLL, Castleman Disease)

Conclusion: Mainly Pemphigus is of 2 types

	Defect	Site of separation	Bullae	Easily Rupture
Pemphigus Foliaceus	Dsg1	st. granulosum (sub corneal)	Superficial	✓
Pemphigus Vulgaris	Dsg3	st. spinosum (supra basal)	Deep.	X

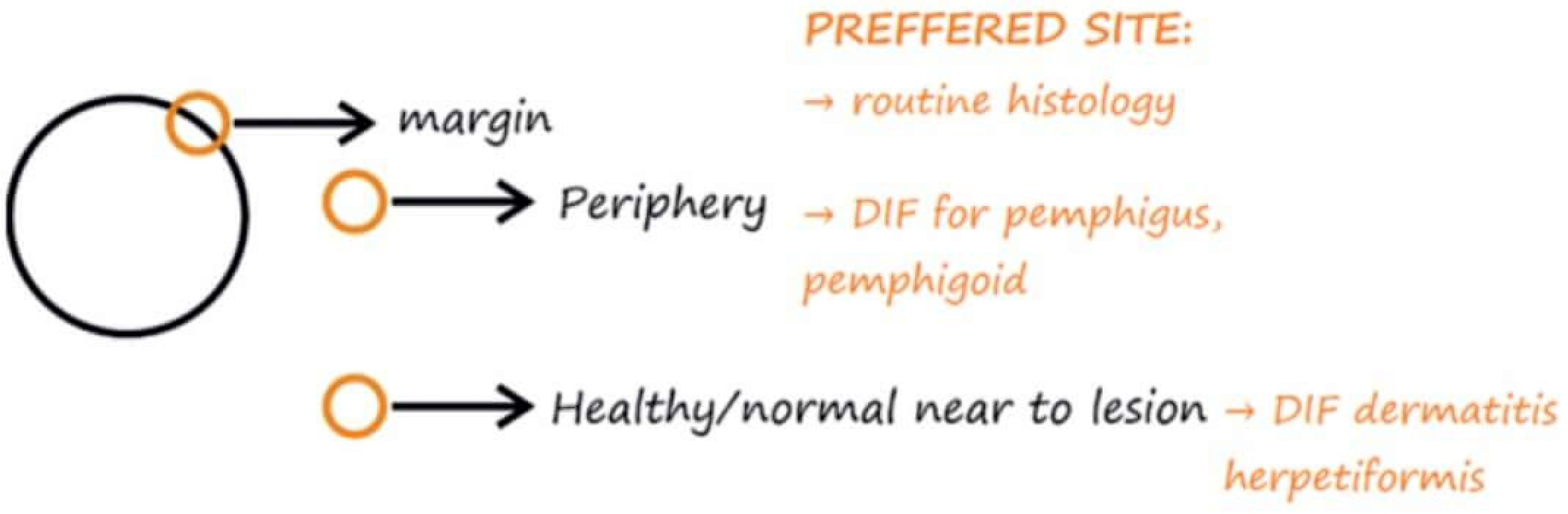
“F comes 1st”

	Pemphigus Vulgaris	Pemphigus Foliaceous
Association: -	HLA – DRO4	Myasthenia gravis, Thymoma
Clinical Features: -	Early Dsg-3 (Buccal mucosa) Late Dsg 3+ Dsg 1 (skin) Buccal Mucosa 	skin 

Involvement of	a. Buccal mucosa: present b. Skin-Bullae: flaccid, suprabasal ↓ May cause painful erosion	Absent Bullae: flaccid, subcorneal ↓ Shallow erosion (MC) with Crusting + scaling.
Sites: -	Buccal mucosa> Scalp, Extremities, Trunk> Periungual	Seborrhoeic distribution
Clinical signs: -	I. Nikolsky Sign – present  PV >PF ii. Bullae spread- sign/ Asboehansen sign- present  PV >PF	Present Present
Investigation: -	I. Histology Acanthocytes - present Row of Tombstone appearance- present ii. Direct Immunofluorescence (DIF) Fish net like pattern of Ig G Ab- Present against Dsg 3>Dsg 1	Absent Absent Present against Dsg 1
Treatment:	a. Immunosuppressive i. Steroids→Dexamethasone ii. Azathioprine iii. Cyclophosphamide Since patient need higher dose for longer duration of treatment. Therefore, Do DCP. DCP → Dexamethasone cyclophosphamide pulse generally, for severe condition 1st ,2nd ,3rd day → high dose (Admitted) next 27 days → low dose (At home) } repeat this for 9 pulse in 1st phase. b. Biological drugs- Rituximab (anti CD_{20}) NHL patient: 375mg/m^2BSA RA patient: 1 gm } slow IV infusion c. IV Ig d. Plasmapheresis } for severe cases	

Important Facts: -

1. **Site of biopsy**


PREFFERED SITE:
margin
→ routine histology
Periphery
→ DIF for pemphigus, pemphigoid
Healthy/normal near to lesion
→ DIF dermatitis herpetiformis

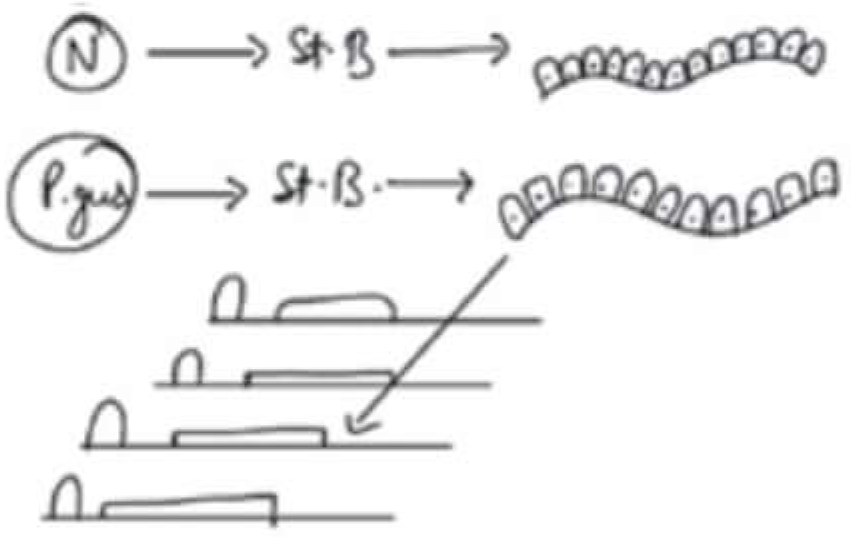
2. **Row of Tomb Stone appearance**

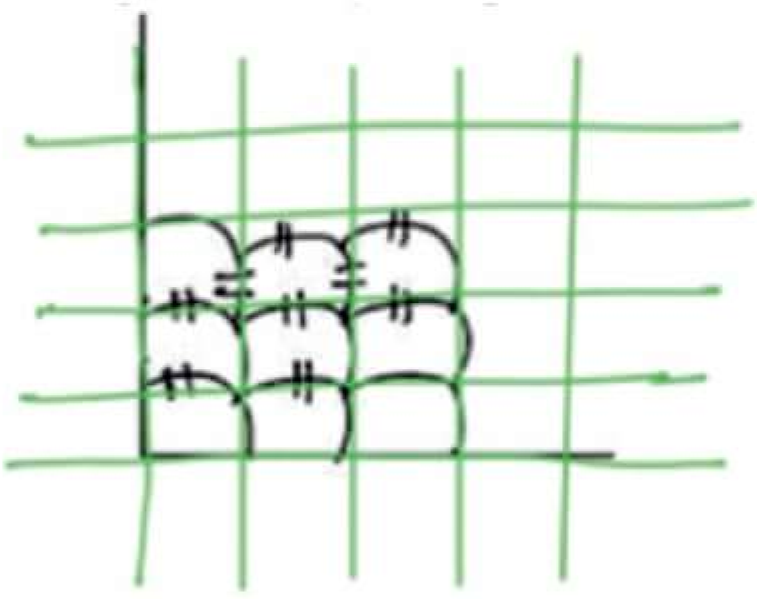
The keratinocytes have gap in between because in pemphigus there is involvement of desmoglein and function of desmoglein is adhesion since adhesion is lost these keratinocytes start separating.

Keratinocytes present in linear fashion having gaps in between gives resemblance to a tombstone

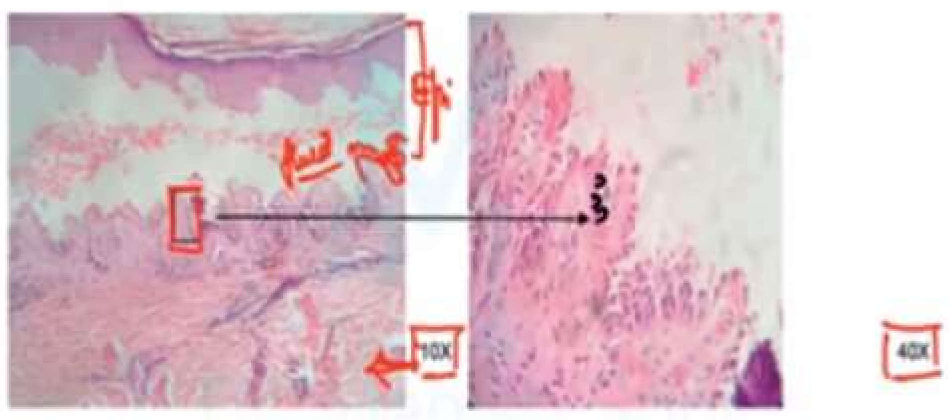
→ Keratinocytes which are still not separated but pemphigus antibodies are formed against desmoglein. Since these desmoglein are present in between the keratinocytes have antibody, they also go and deposit there in between & give an appearance like fish net on DIF.

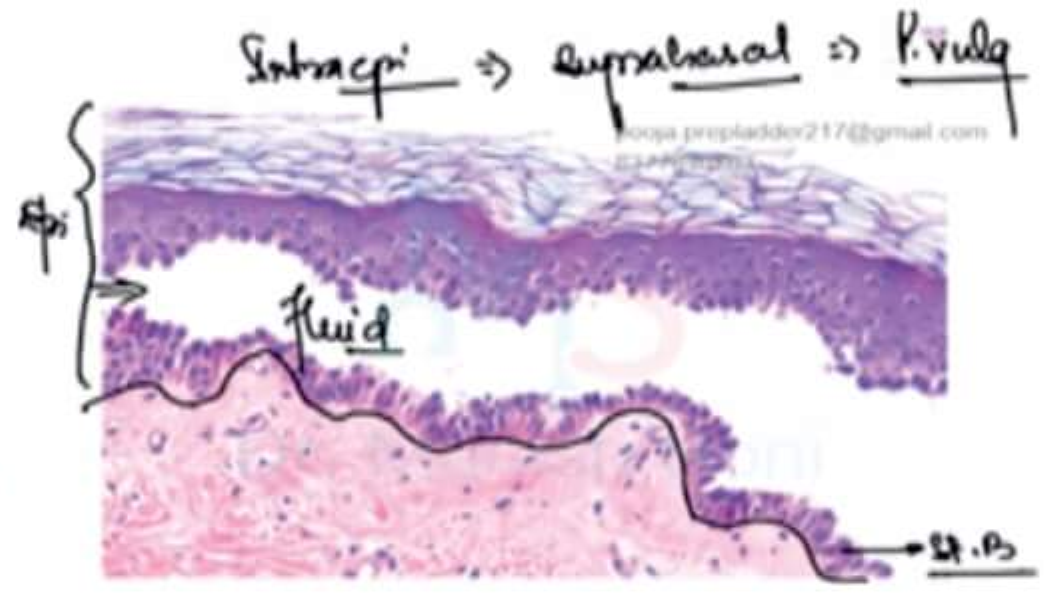
Fish net like pattern is due to intracellular deposition of Anti desmoglein antibody (Anti Dsg Absent)



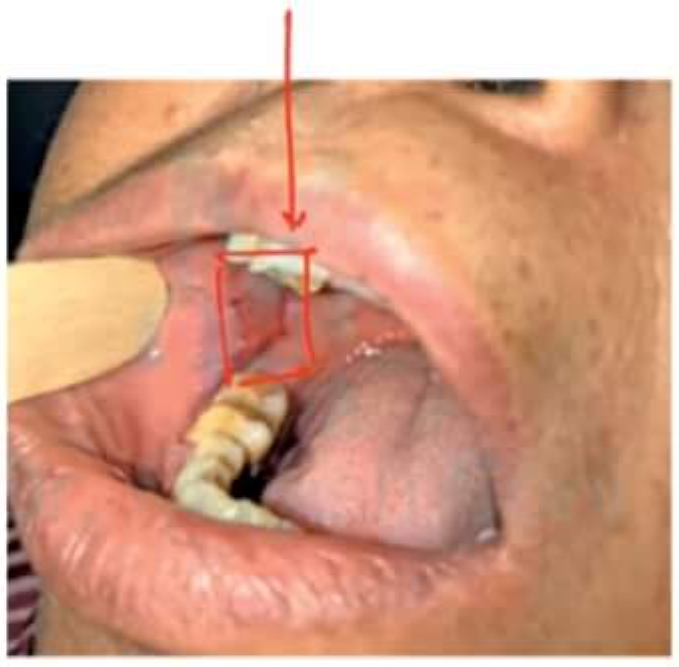


ROW OF TOMBSTONES





PEMPHIGUS VULGARIS

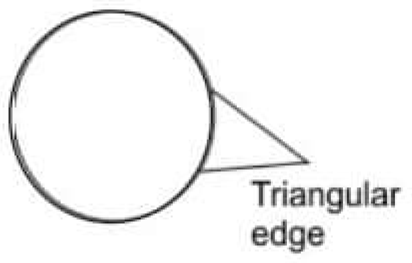
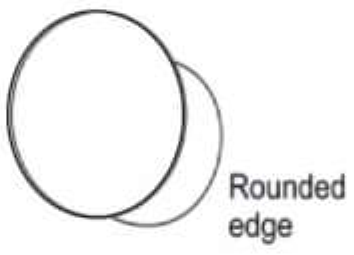


B. SUBEPIDERMAL BLISTERING DISORDERS

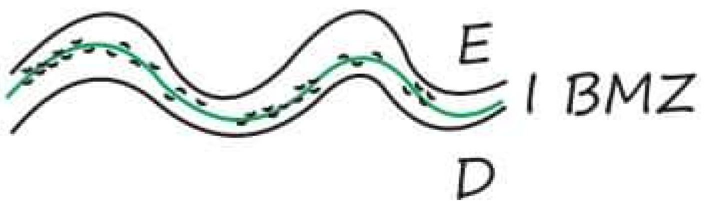
1. Bullous Pemphigoid

Is also an Immunobullous disorder, Antibodies are formed against BP Ag 2> BP Ag 1 leading to subepidermal bullae.

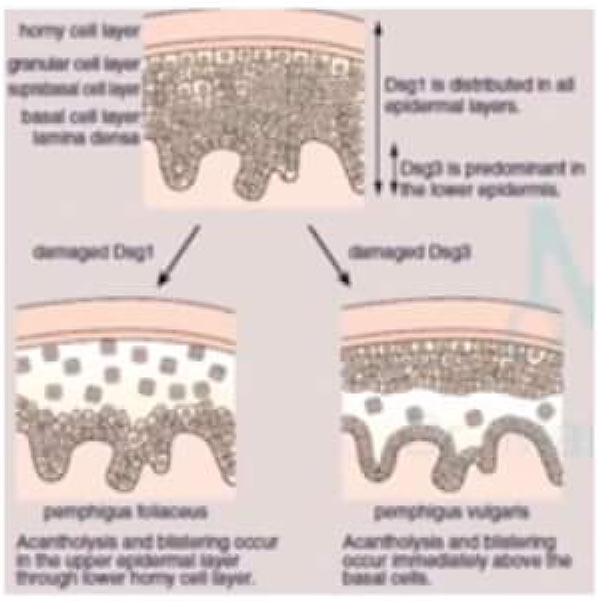
→ These subepidermal bullae are associated with symptoms like
- pruritus
- perilesional erythema'
- Milia formation (occurs At site of healing)
  - → (Are keratin cyst which are Denovo formed over face)

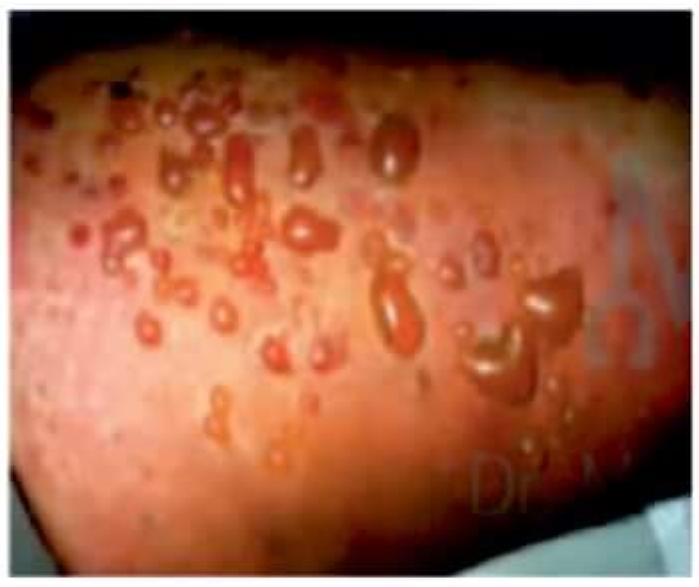
	Pemphigus	Pemphigoid
Age of onset	40-60	60-80
Lesions	Flaccid bullae, painful erosions	Tense Bullae
Mucosal Involvement M Intraepidermal Bullae I Nilkolsky sign N Acanthocyte A Row of tombstone R	Present	Absent
Bullae spread sign	Present 	Present 

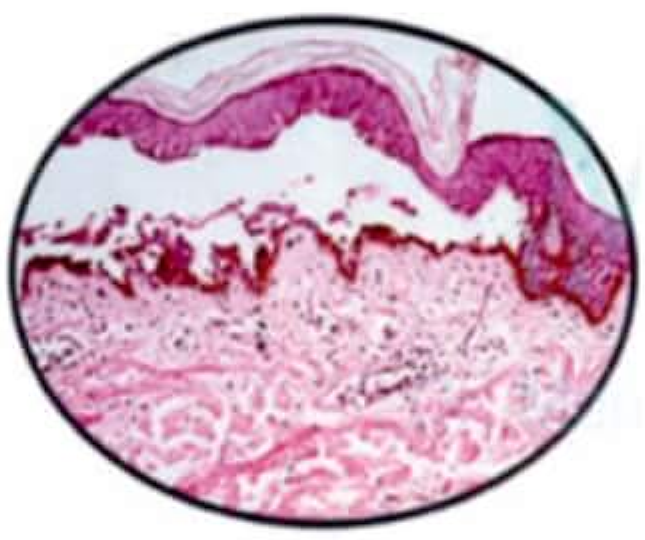
Investigation
1. Histology: Subepidermal split with collection of eosinophil's
2. DIF: linear IgG & C_3 deposit along BM zone.

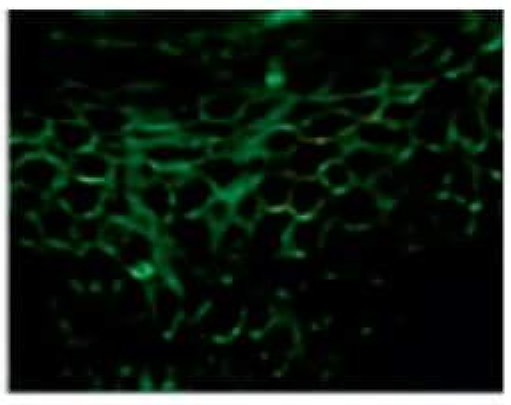


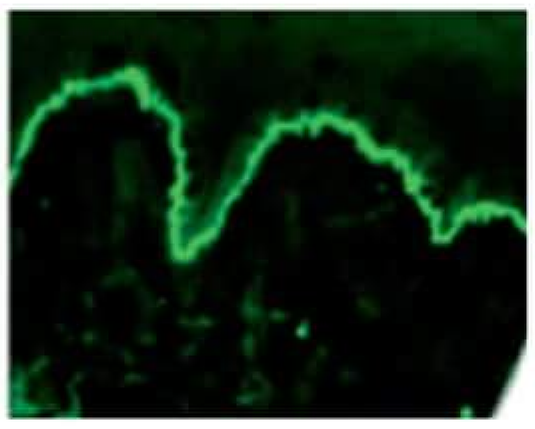
Treatment
1. Tetracyclines (anti-inflammatory)
2. Steroids
3. Dapsone


horny cell layer
granular cell layer
suprabasal cell layer
basal cell layer
lamina densa
Dsg1 is distributed in all epidermal layers.
Dsg3 is predominant in the lower epidermis.
damaged Dsg1
damaged Dsg3
pemphigus foliaceus
Acantholysis and blistering occur in the upper epidermal layer through lower horny cell layer.
pemphigus vulgaris
Acantholysis and blistering occur immediately above the basal cells.



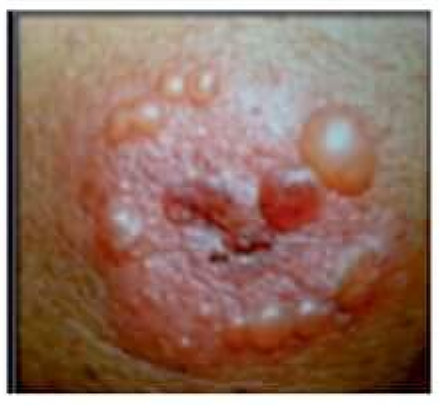




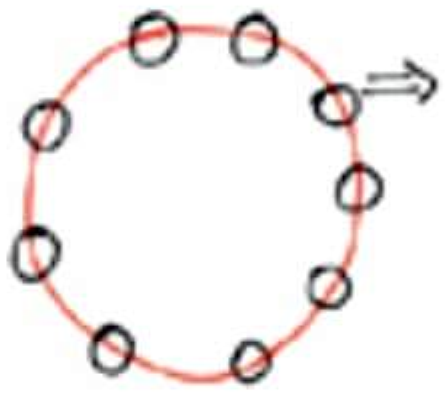


2. Linear IgA disease

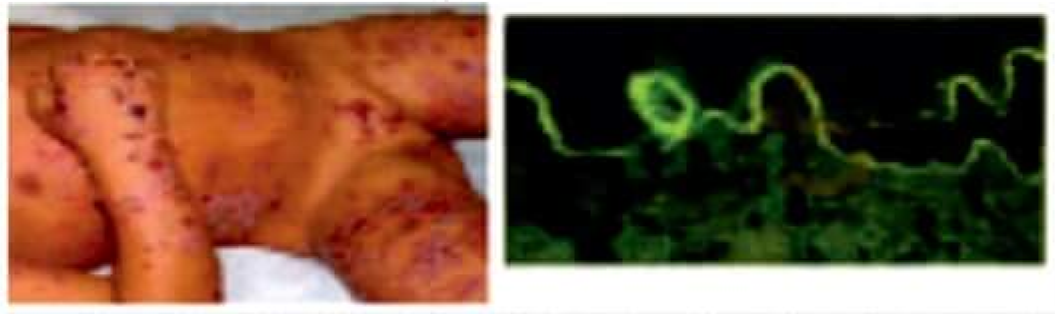
- Immunobullous disorder, Antibodies are formed against mainly BP Ag 2 >BP Ag 1.
- Clinically, has bimodal presentation
  - occurs in children <5 years Involving face (perioral area),Groin (perianal area)
  - Occurs in adults generally 60 years. Involving Trunk & Extremities



→ DOC: Dapsone



Linear IgA disease



- Clinical Features: Erythematous urticated plaque around it grouped fluid filled vesicle develop giving Characteristic appearance "String of pearl/ cluster of jewel"

Investigation:

DIF: linear^{Q} IgA deposits seen along BMZ

Treatment:

DOC- Dapsone 1-2 mg/kg/dwt.

3. Dermatitis Herpetiformis/Duhring's Disease

It occurs due to gluten HSN & associated with celiac disease.

↓

Is type of gluten enteropathy on intestinal biopsy

↓

Villus atrophy

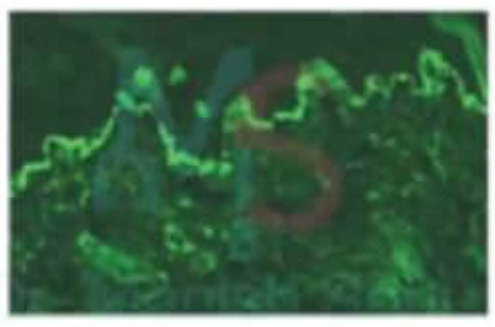
Other association

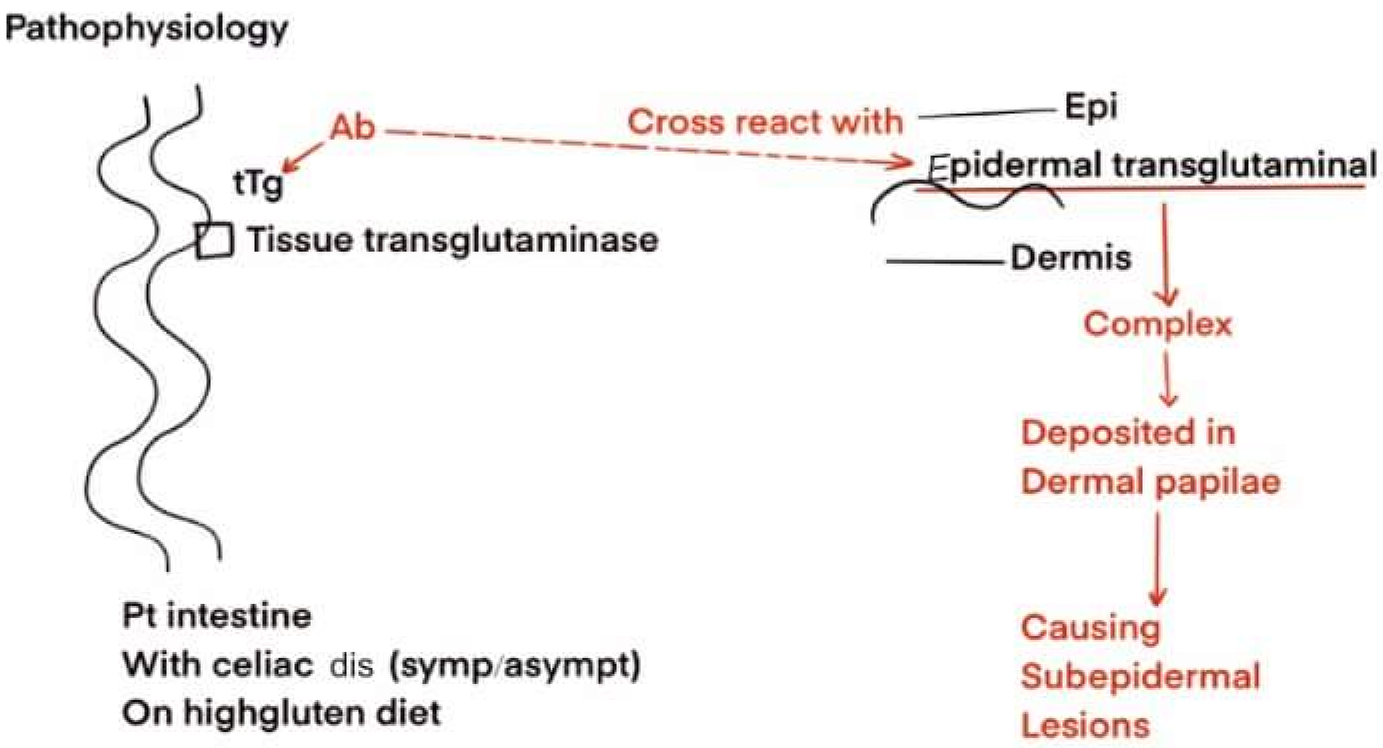
→ HLA DQ2, HLA B8^{Q} JIPMER 18

→ Anti endomyseal AB

→ Anti-transglutaminase AB

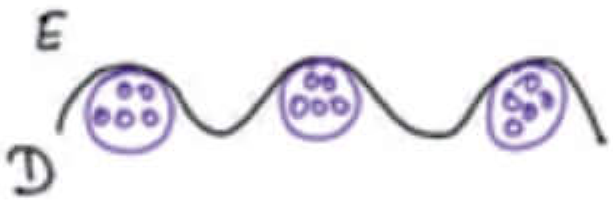
Pathophysiology:





Clinically:

- extremely pruritic (moderate-severe)
- vesicopapules over extensors MC (Elbow, knee, back, buttock)
- MC presentation in Excoriated lesion



Investigation:

1. **Histology**- papillary Tip micro abscess
   Small collection of neutrophils in papillary Tip
2. **DIF:** granular^{Q} IgA deposits in dermal papillae.

Treatment:

TOC- gluten free diet

Avoid “Brow”	Given
→ Barley	→ Rice
→ Rye	→ Maize
→ oat	
→ wheat	

Q. Which of the following is given in patient with celiac disease.

a. Barley
b. Rye
c. Oats
d. Wheat

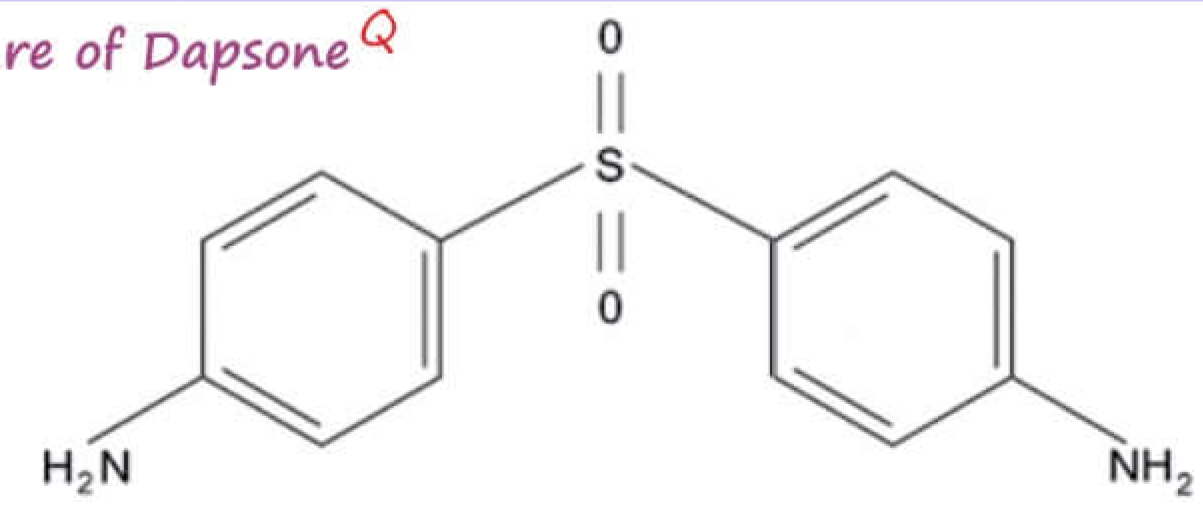
Answer- Oats (as oat has least amount of gluten)

DOC- Dapsone (therapeutic + diagnostic)

- almost all case resolves within 7 days
- Relapses can occurs, as Ab level still persists giving symptoms relief.
- Therefore, Its DOC not TOC.

If question mentions that patient Ab level ↓ indicates person is on gluten free diet.

Structure of Dapsone^{Q}

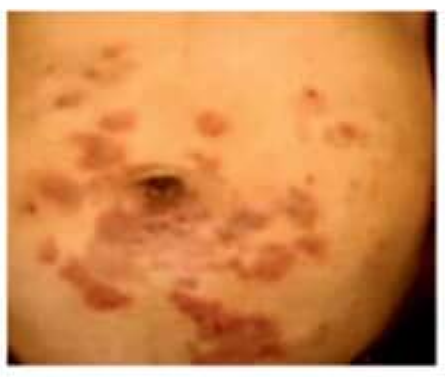


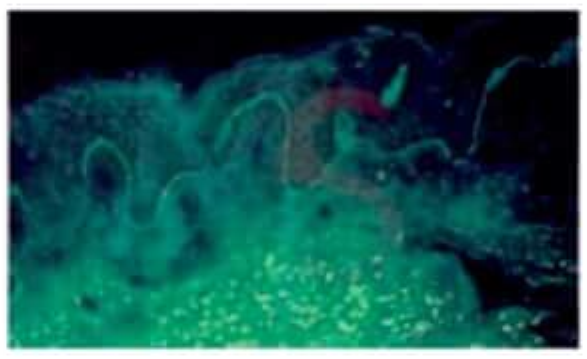
4. Herpes **(misnomer)** gestationalis/pemphigoid gestationalis

- It is also an autoimmune condition where Ab are formed against BP Ag 2 >BP Ag 1.
- Clinically presents in 2nd/ 3rd trimester of pregnancy.

Clinical Features

- lesions are periumbical
- vesicular eruptions progress & involve whole abdomen
- spontaneously regress
- fetal mortality ≈30%





Investigation:

1. DIF: linear C_3> IgG deposits along BMZ

Treatment:

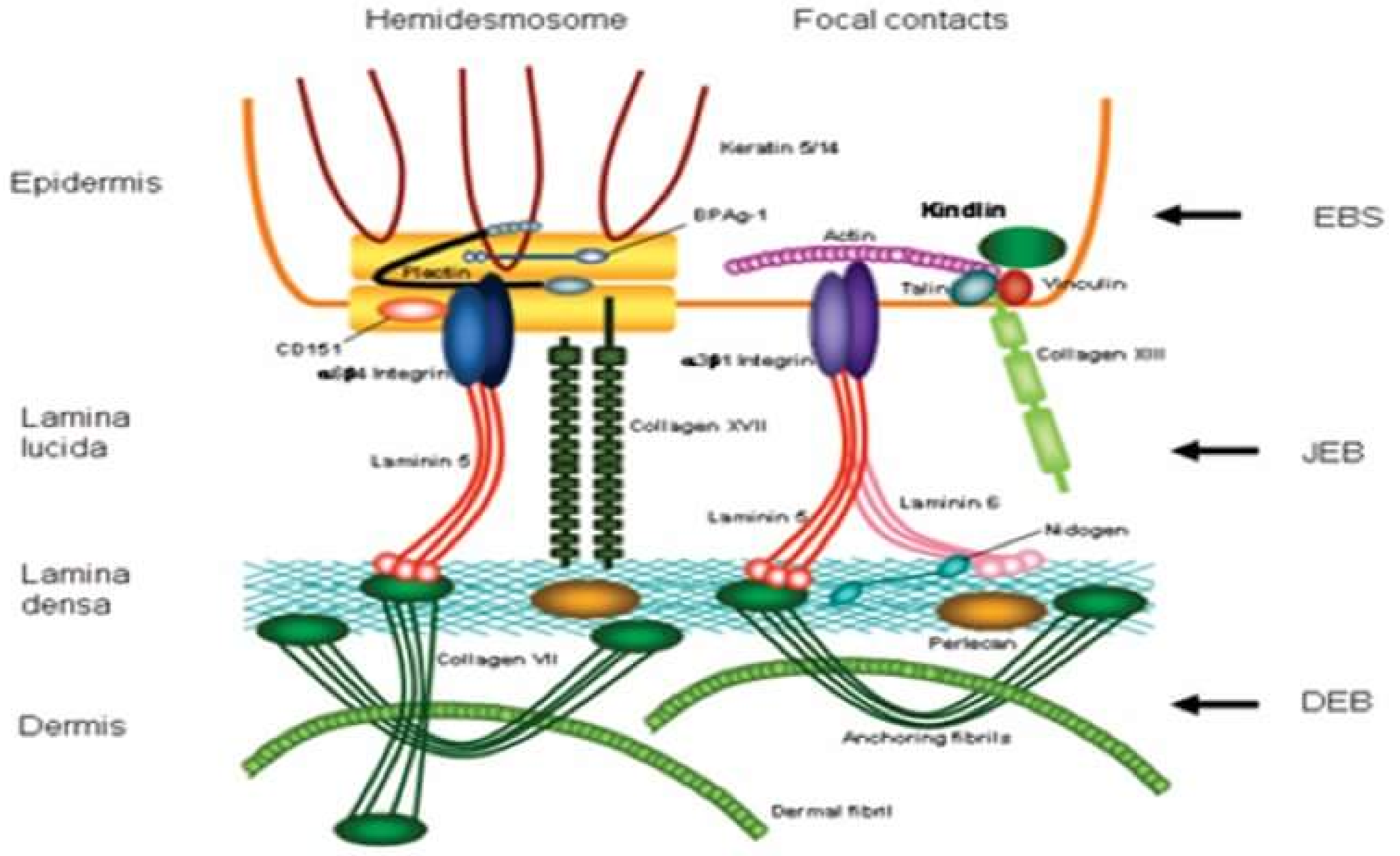
DOC- systemic corticosteroid (prednisolone)

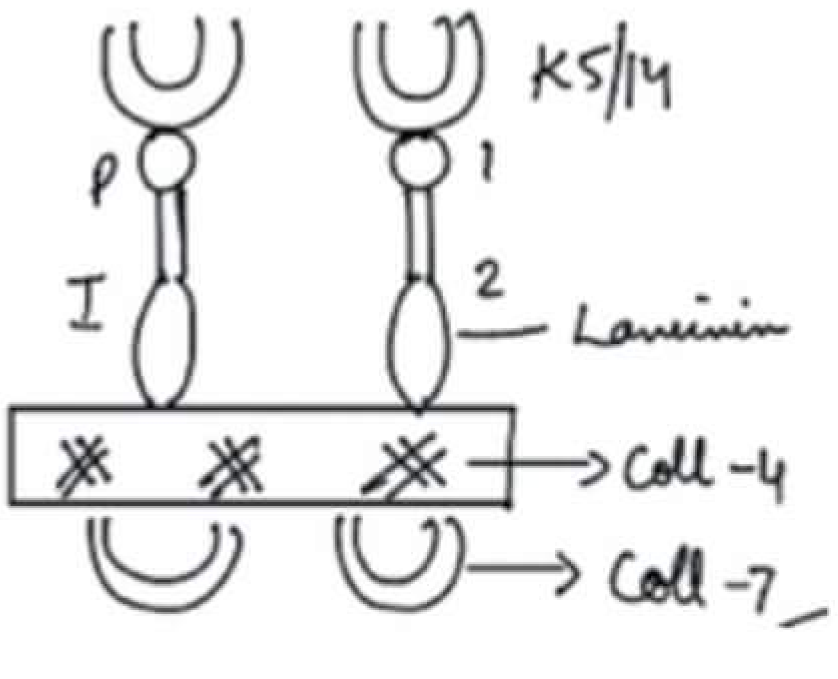
C. Epidermolysis Bullosa/ Mechano Bullous Disorder

- These bullae are generally seen at site of Trauma/pressure handling/friction.
- Clinically Bullae Can be both intraepidermal /subepidermal

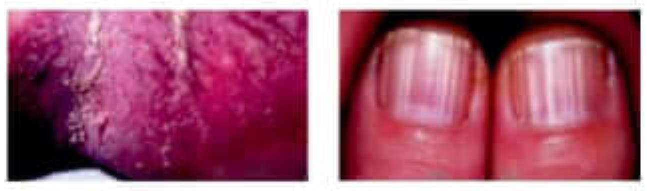
2 Types

Congenita	acquisita
- congenital - early onset - more common - genetic condition	- acquired - late onset - rare - Immuno-bullous condition
Depending on genetic defect	
1. E.B simplex- K5/14 Defect ^{Q} 2. E.B junctional- laminin defect^{Q} 3. E.B dystrophicans- collagen 7 defect^{Q}	Ab are formed against Non Collagenous domain of collagen 7
DIF negative no role	DIF – positive

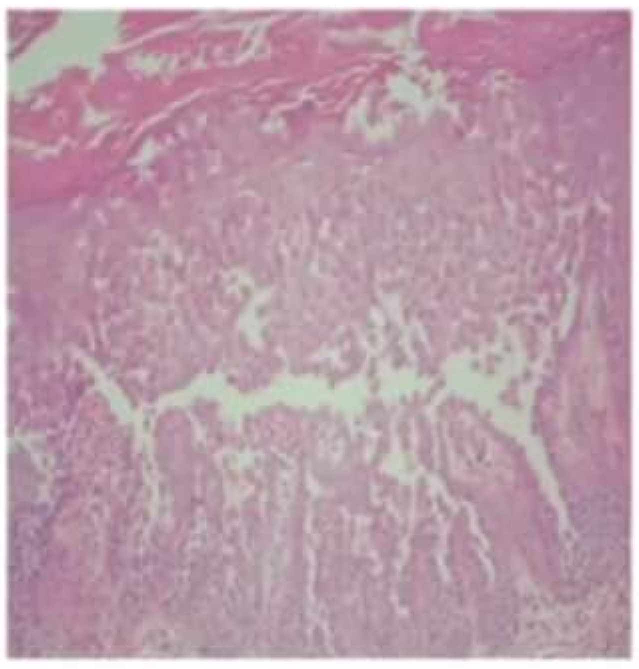


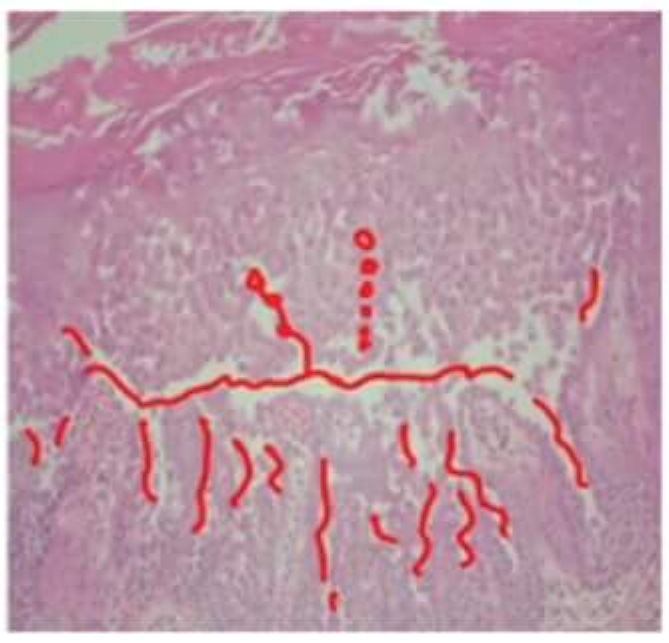


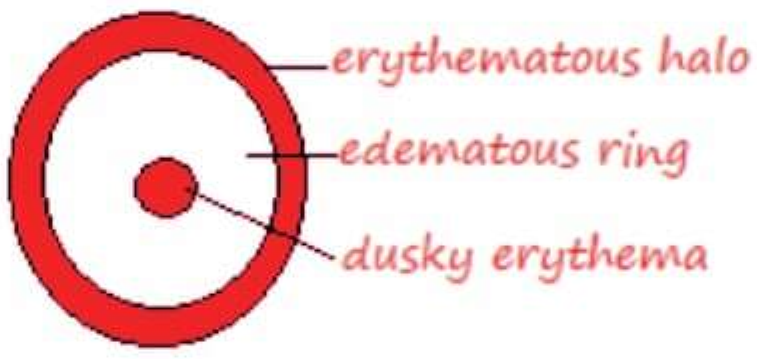
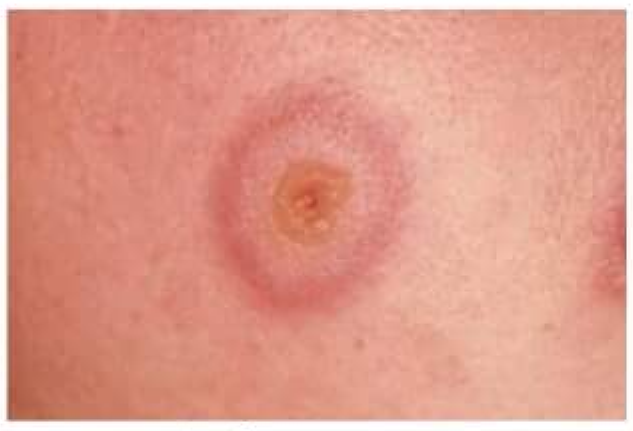
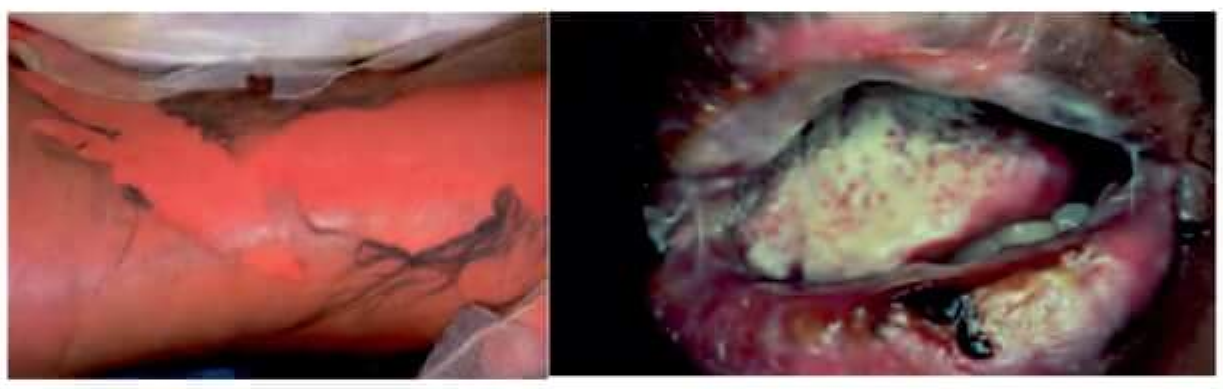
EB Type	EB subtype	Protein/gene defect	Inheritance
EBS	a. Weber Cockayne EBS	K5,K14	AD
JEB	a. Herlitz b. Non Herlitz	Laminin 5 Laminin 5 Type XVII collagen	} AR
DEB	a. Dominant DEB b. Hallopeau siemens RDEB c. Non hallopeau siemens RDEB.	Type VII collagen Type VII collagen Type VII collagen	AD } AR

	D. Darriers disesase	**E. Hailey hailey disease /Chronic Benign familial Pemphigus –**
	these are inherited acantholytic disorder.	
Inheritance:	AD	AD
Defect:	Ca^{2+} Atpase involvement	Ca^{2+} Atpase involvement
Gene defect:	ATP 2 A_2 involvement	ATP $2C_1$ involvement
	Encode for SERCA	
Clinical Features:	1. Lesions verrucous, warty greasy, sand Paper like feel 2. Site: Seborrheic areas 3. Nails: V shaped splitting over Distal nail plate (draw) 4. Investigation Histo: Acanthocytes, Dyskeratosis Corps grain, Corps bonds	recurrent flaccid, vesicular eruption Intertriginous areas (skin folds) longitudinal white bands on nail  Acanthocytes, Dyskeratosis Dilapidated brick wall appearance
		CBFP (old nomenclature) ↓ – as recurrent – as localised in few place – as family H/o treatment – grouped under pemphigus. Microscopic examination reveals many layers of detached Keratinocytes (acantholysis) giving appearance similar to a dilapidated brick wall.

Microscopic examination reveals many layers of detached Keratinocytes (acantholysis) giving appearance similar to a dilapidated brick wall.
Seen in HHD/CBFP





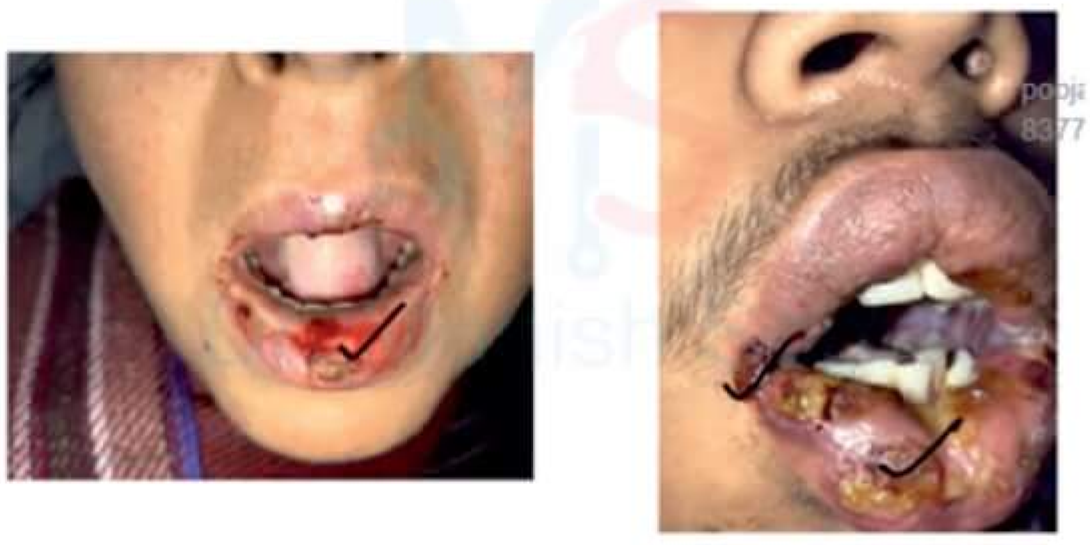
F. Erythema multiforme	**G. Epidermal Necrosis**
Etiology: Known - viral infection HSV> mycoplasma - Drugs Unknown	MC: Drugs
Clinical Features: Target lesions/Bulls eye/ iris Generally over distal extremities	**Clinical Features** Targetoid lesions (2 rings) ↓ Later cause epidermal separation Depending on BSA <10% k/A SJS 10-30% K/A overlap >30% K/A TEN
  **Erythema multiforme(TARGET LESIONS)**	
Mucosal involvement is absent [if present K/a epidermal multiforme Major]	Mucosal involvement always present. inform of haemorrhagic crust with epidermal necrosis
Nikolsky sign negative	Nikolsky sign positive (Pseudonikolsky) As due to epidermal necrosis not due to Acantholysis.

Treatment:

1. Avoid offending agents
2. Conservative treatment fluid & electrolyte
3. Progressive/severe: treatment with corticosteroid ,cyclosporine.

SJS/TEN

Hemorrhagic crusting



Important facts:

→ Carbamazepine HLA B 1502 Q. JIPMER
Association with SJS/TEN

→ Scoring systems

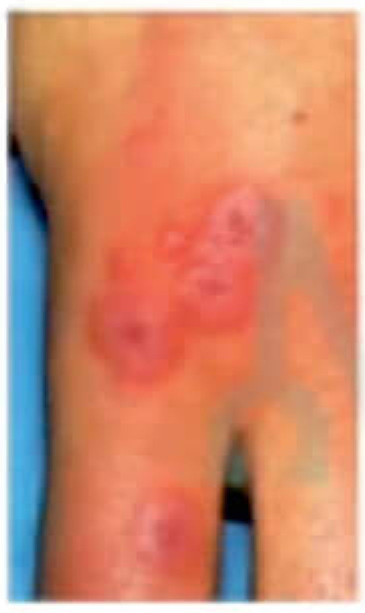
- SCORTEN → TEN patients
- SCORAD → Atopic dermatitis
- PASI → Psoriasis area severity index
- VASI → Vitiligo area severity index
- MASI → Melasma area severity index
- CASPAR → psoriatic arthritis

Q. 35 years old male developed flaccid vesico-pustules with crusted erosions and expanding circinate plaques over axillae associated with malodorous discharge and fissures (rhagades). Asymptomatic longitudinal white bands were present in the nails.

A. Candida intertrigo with onychomycosis.
B. Tinea corporis with tinea unguium
C. Darier's disease and its nail changes
D. Hailey- hailey disease and its nail changes

Ans- D

Q. The patient came with h/o lesions distribute preferentially on the distal extremities. What is the probable triggering factor?



A. Bacterial infection
B. Viral infection
C. Fungal infection
D. Drug

Ans- C) Viral infection

Loss-of-function mutations in the ATP2 A2 gene encoding sarco/endoplasmic reticulum calcium adenosine triphosphates inform 2 (SERCA2). Which impair intracellular Ca^{2+} singling Multiple discrete scaling of crusted, pruritic papules. Confluence to large plaques covered by hypertrophic warty masses. Distribution corresponding to the "Seborrheic area" chest, back, ears, nasolabial folds, forehead, scalp Nails thin, splitting distally and showing characteristic V-shaped scalloping

PIGMENTARY DISORDER

A) HyperpigmentoryDisorder

→ Are disorders where excessive melanin is present mostly in epidermis, sometimes dermis as well.

→ **Two ways to differentiate epidermal melanin from dermal melanin**:

a) **color of lesions** → epidermal is brown/ black in color
→ dermal melanin is blue

b) **wood's lamp examination** → blue color of epidermis accentuates under woods lamp examination
→ blue color of dermal melanin is due to tyndall/optical effect.

1. Melasma:

Etiology: → Hormonal: Pregnancy, OCP intake and hormonal replacement therapy
→ Sun exposure: excessive UVB exposure
→ Associated with hypothyroidism

Clinical Feature: - Brownish Discoloration, can be at various sites
↓
Due to excessive melanogenesis

Depending on sites, type of melasma are:

I. Centrofacial (MC)
II. Mandibular
III. Maxillary

Site: - Malar, nose, forehead

Treatment: - Tyrosinase inhibitors/depigmentary agents (Tyrosinase as a Rate limiting enzyme for Melanogenesis)

E.g.: → Topical
→ Hydroquinone
→ Azelaic acid
→ Kojic acid
→ Arbutin

Klingmans formula: Hydroquinone + Tretinoin+ steroids

Advise: Sun Protection-physical-cover the face
Chemical-Suns cream

Important fact:

1. Exogenous ochronosis- is the bluish grey- grey discoloration of the skin due to excessive topical application of hydroquinone.

2. contact leukoderma- skin becomes white due to contact with chemicals

e.g. a. Monobenzyl ether of Hydroquinone:
Present in Rubber, is most potent depigmentary agent

Rubber shaped

b. Para 3° butyl phenol: Present in Adhesive in bindi

c. Para 3° butyl catechol: Present in Hair dye

3. UV spectrum– the range of UV radiation is 200-400nm

UVC- 200-290nm wavelength, wavelength 290nm is cut by ozone layer-0%, also known as germicidal

UVB- 290-320nm wavelength- 5%, also known as sun burn spectrum

UVA- 320- 400nm wavelength- 95%, (UVA I- 340-400nm, UVA II- 320-340nm)

2. Ceruloderma

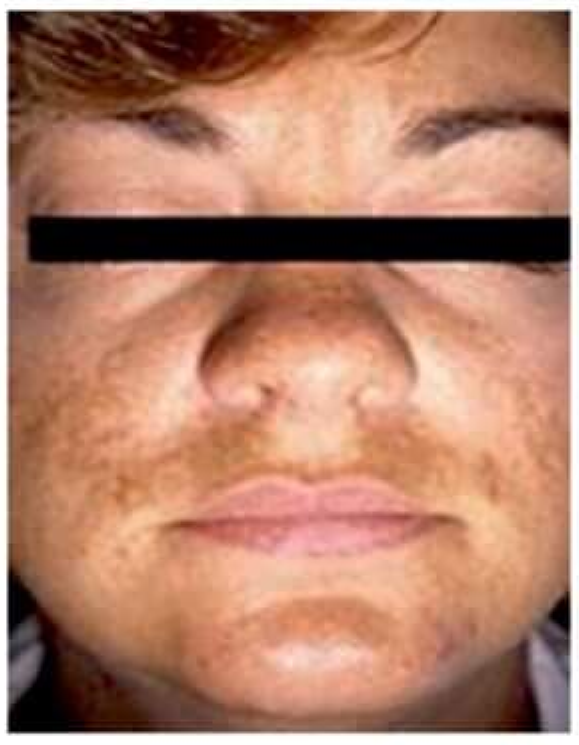
↓

Blue color lesion is seen

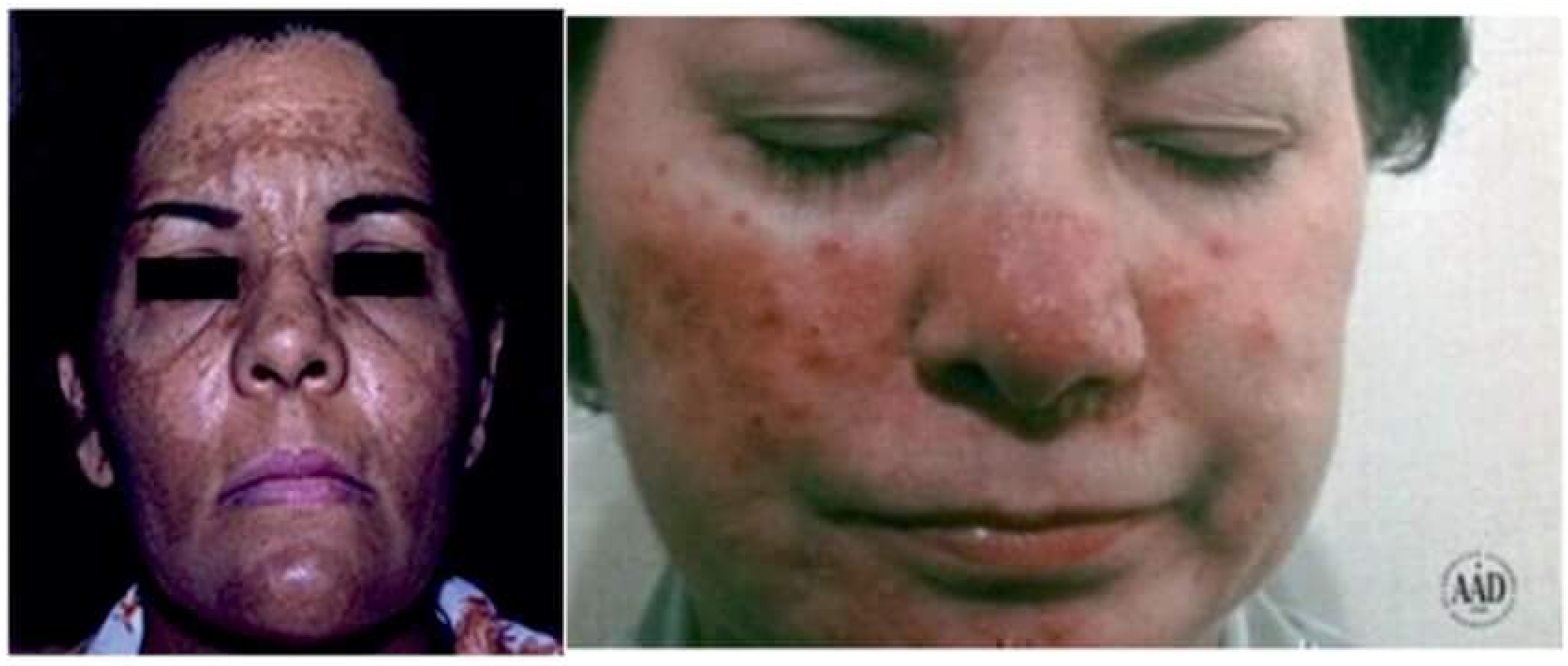
It is the condition when melanin present in dermis

Eg 1. **Mongolian spot**- bluish discoloration in infants mc site-lower back infant, which spontaneously resolves

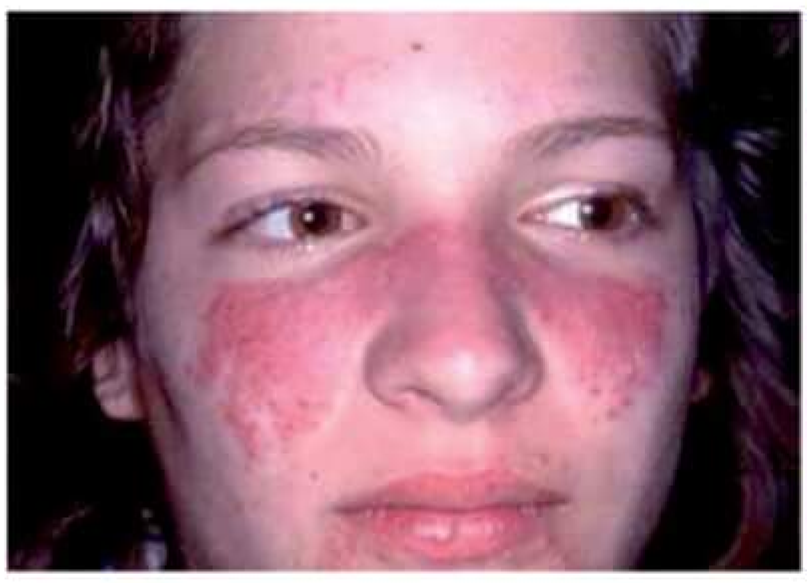
Both
- 2. **Nevus of OTA**→ Along ophthalmic & maxillary division of trigeminal nerve
- 3. **Nevus of ITO**→ Along Acromioclavicular Nerve (posterior supraclavicular nerve and lateral branch of cutaneous nerve
  - → unilateral
  - → bluish grey discoloration
  - → treatment: Ruby (Q-switched mode) laser or Nd: YAG laser



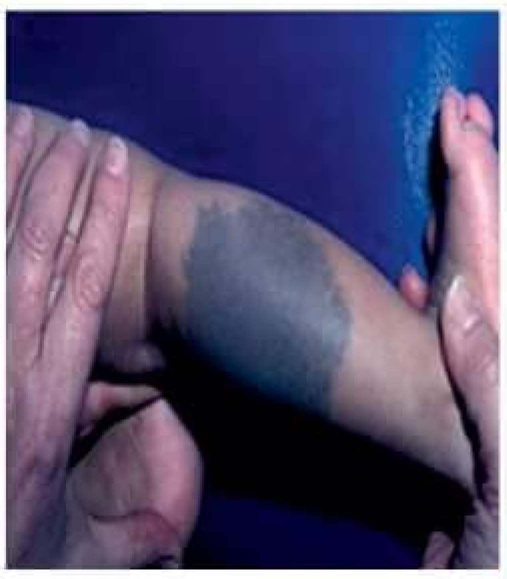
Melasma

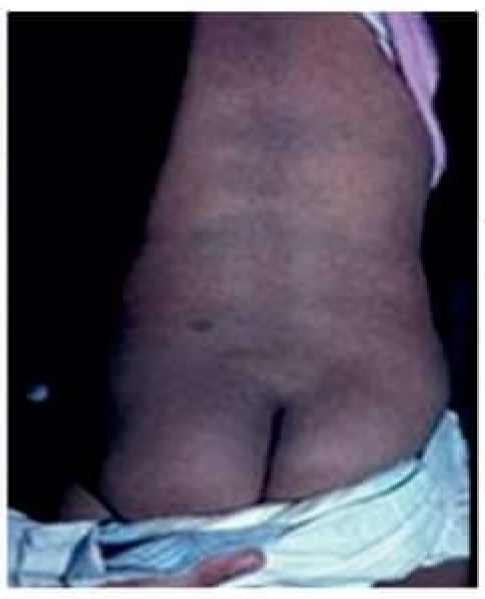


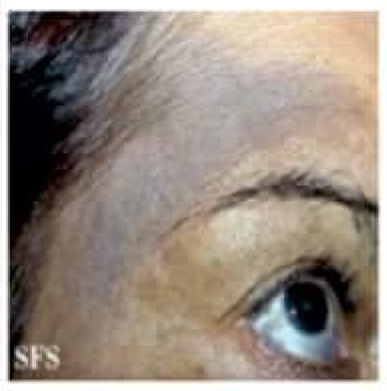
Rosacea involvement of nasolabial fold)



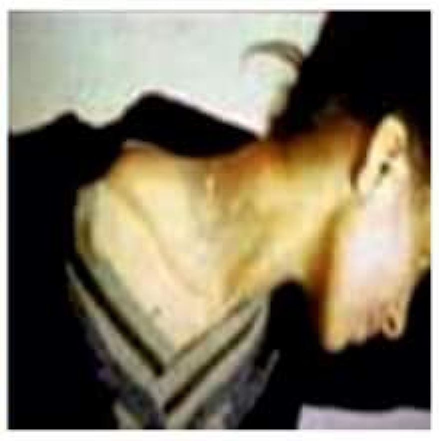
Nasolabial sparing in SLE







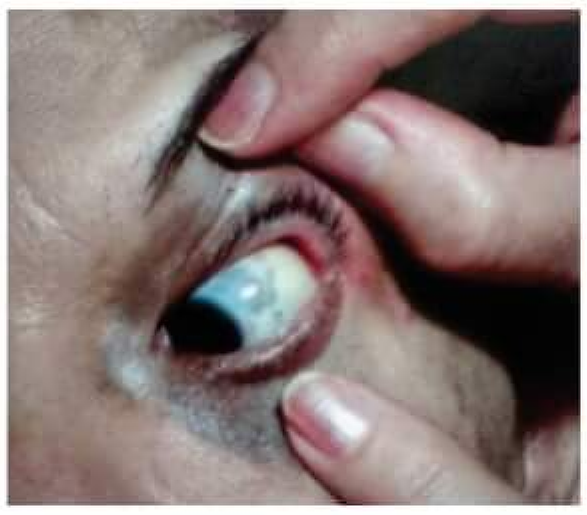
Mongolian spotnevus of OTA



nevus of ITO

Rosacea and SLE are differential diagnosis of each other, but Melasma is not as melasma lesions are brown and in rosacea lesions are red.

Q. A lady came with complaints of a bluish lesion over left side of forehead and left eye. Shows irregular bluish lesion in left superior conjunctive and forehead. Diagnosis?



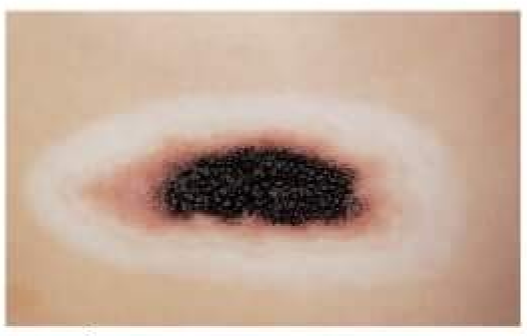
A. Nevus of Ota
B. Nevus of Ito
C. Becker's nevus
D. Mongolain spot

Ans- A)

Important facts

1. Becker's Nevus: -

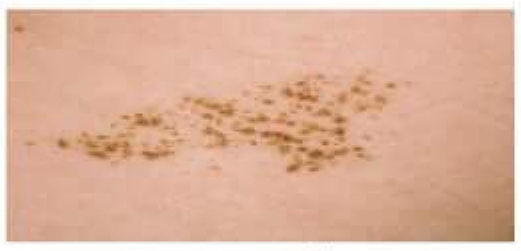
- Hyperpigmented patch with Hypertrichosis over upper trunk
- Usually develop during adolescence in Males



Halo nevus/ Suttons nevus

2. Halo nevus/ Suttons nevus

- Benign melanocytic nevus surrounded by depigmented halo



Nevus spilus

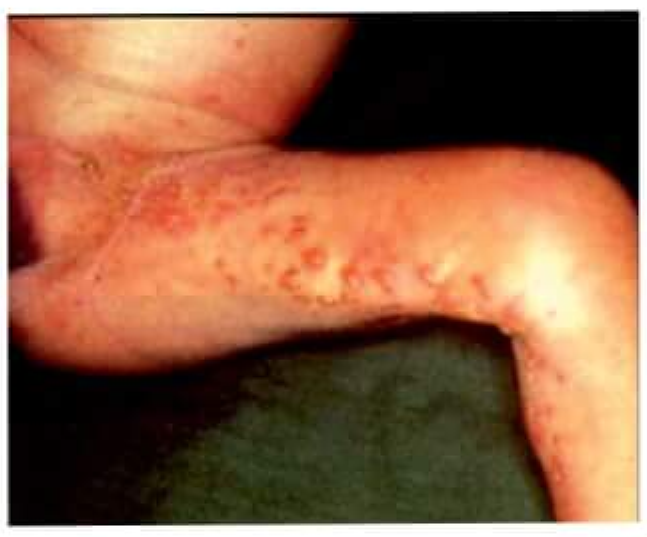
3. Nevus spilus

- Also known as speckled lentiginous nevus
- Presents as hyperpigmented circumscribed macules, usually more darkly pigmented "spot"

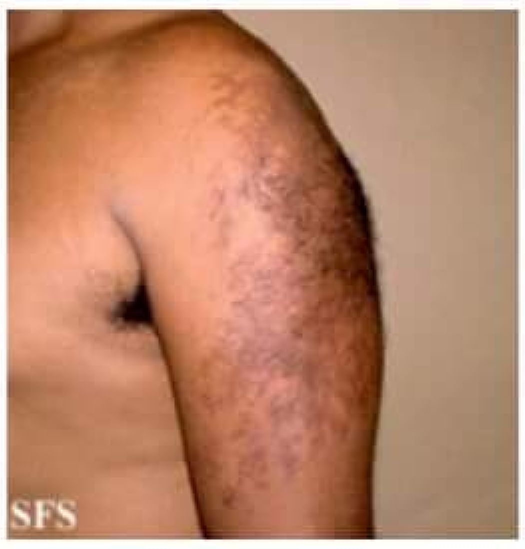
4. Incontinentia Pigmenti

- X-linked dominant Condition[Q]
- patient presents with developmental defects of eyes, teeth, CNS with cutaneous lesions
- lesions follow imaginary lines of development in a linear fashion known as lines of blaschko.
- 4 clinical stages:

Inflammatory macules, papules, vesicles and pustules
↓
Hyperkeratotic &**Verrucous** lesions
↓
Grey- brown **Hyperpigmentation**
↓
Atrophic, **Hypopigmented** and depigmented



Incontinentiapigmenti



becker's nevus

B. HYPOPIGMENTRY DISORDERS: -

1. **Vitiligo** → Autoimmune condition (MC) → in few cases family history is positive (15%)

Types

Segmental
- Unilateral
- **Along a dermatome**
- **Neurogenic hypothesis**
  Symmetric

non-segmental

Localized
- In mucosa – mucosal
- In small area – Focal

Generalized
- **Vitiligo Vulgaris**: Most common B/L
- **Acrofacial:** Hand, feet and Face involved
  Lip tip type variant of acrofacial In this involvement of lip along with the tip of penis or nipples.
- **Vitiligo universalis**: associated with autoimmune disorders like Hashimoto thyroiditis, Diabetes Mellitus, pernicious anemia, alopecia areata.

Prognosis: 1. Leukotrichia
2. long standing type
3. lesions over bony prominences – Poor prognosis
4. acrofacial

Treatment: - 1. Topical: Steroid
Calcineurin inhibitors like tacrolimus, pimecrolimus
Vitamin D analogs
2. systemic: steroid
Azathioprine
Levamisole
3. Phototherapy
- PUVA – Psoralanes followed by 'UVA' light
- PUVASOL – Psorolane followed by Sunlight
  ↓
  Solar
- NBUVB(narrow band UVB) – expose lesions to 311 nm wavelength
- Targeted phototherapy- Excimer laser used which emits light of 308nm
- wavelength Psorlanes → MOA bind to DNA
  Types→natural – Eg 8 methoxy psoralen
  Synthetic – eg trimethoxy psorlanes

Route of administration

→ systemic $\xrightarrow{\text{wait for 2hrs}}$ light
→ topical $\xrightarrow{\text{wait for 30 min}}$ light
– give every alternate day exposure to sunlight. preferred time is '11 am to 1pm'

4. Surgical treatment: indicated → if medical treatment fails and disease is stable for >2ys

Modalities: 1. Skin grafting → ultra-thin Partial thin skin graft
→ Punch graft
2. Non cultured autologous melanocytic transfer
3. cultured autologous melanocytic transfer

OTHER HYPOPIMENTARY DISORDERS:

PITYRIASIS ALBA	INDETERMINATE LEPROSY
1. Lesions are single. Generally present over face and common in children.	1. Lesions are single. Generally present over face and common in children
2. **scaling present**	2. **scaling absent**
3. **History of atopy present**	3. **history of atopy absent**
4. **atrophy absent**	4. **atrophy is present**
5. **sensation intact**	5. **sensations are impaired**

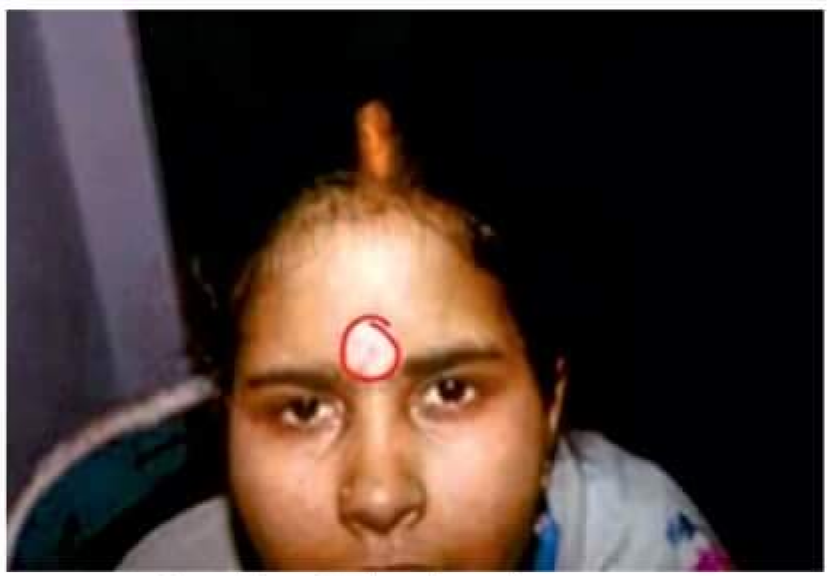
POST KALA AZAR DERMAL LEISHMANIASIS	LEPROSY (LEPROMATOUS)
1. hypopigmented lesions seen over trunk	1.hypopigmented lesions seen on trunk
2. nodular lesions seen on face	2. nodular lesions seen on face
3. endemic in Bihar	3. endemic in Bihar
4. past history of fever is **present**	4. past history of fever is **absent**
5. nerve thickening is **absent**	5. nerve thickening is **present**
6. sensations are **intact**	6. sensations are **impaired**

Important facts:

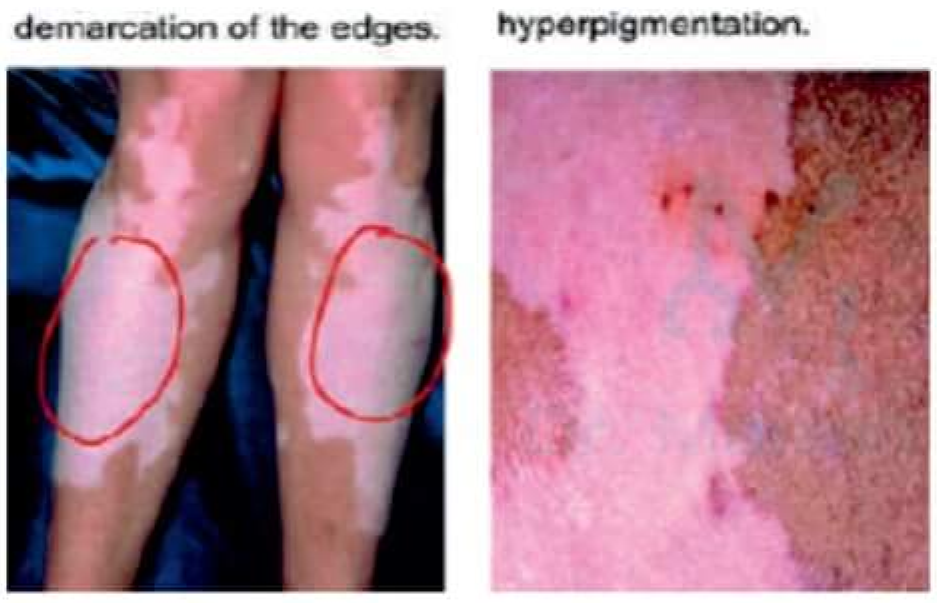
DOC:

- a. **visceral leishmaniasis (kala azar)**– liposomal amphotericin B
- b. **post kala azar dermal leishmaniasis**– Miltefosine

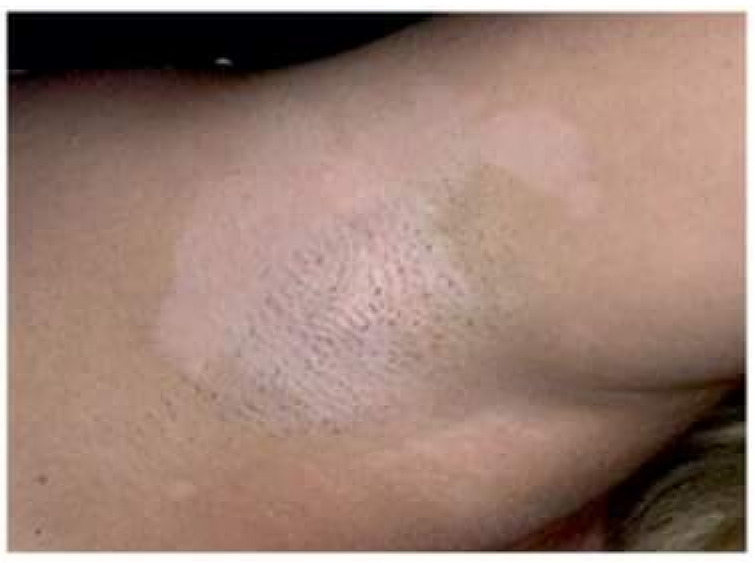
→ pityriasis alba is a self-limiting condition.

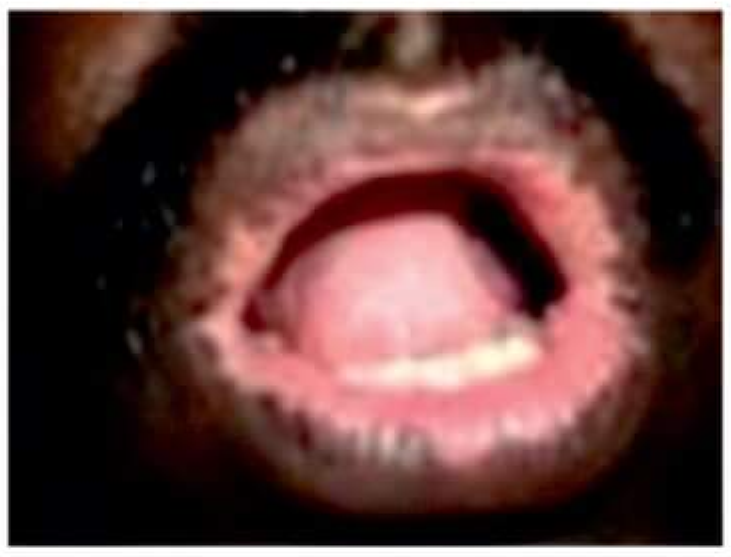


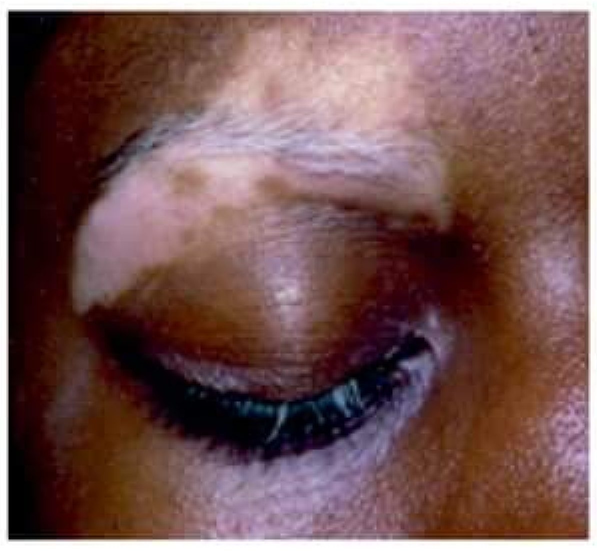
Contact leukoderma

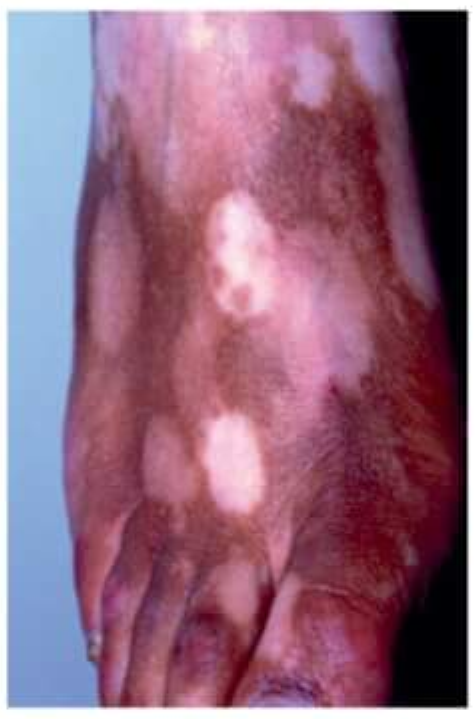


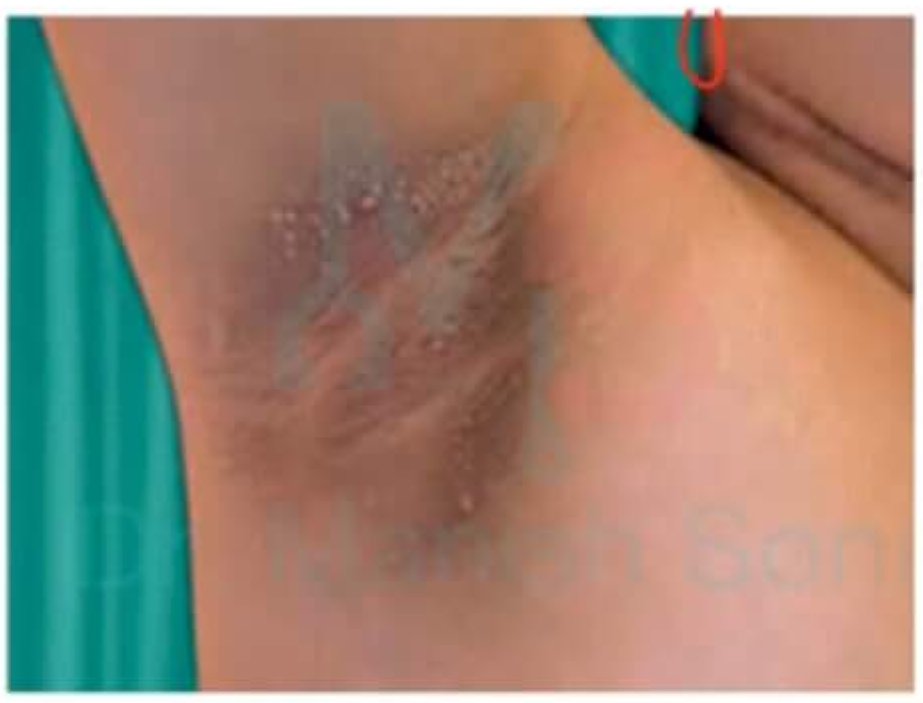
vitiligo vulgaris (mc)

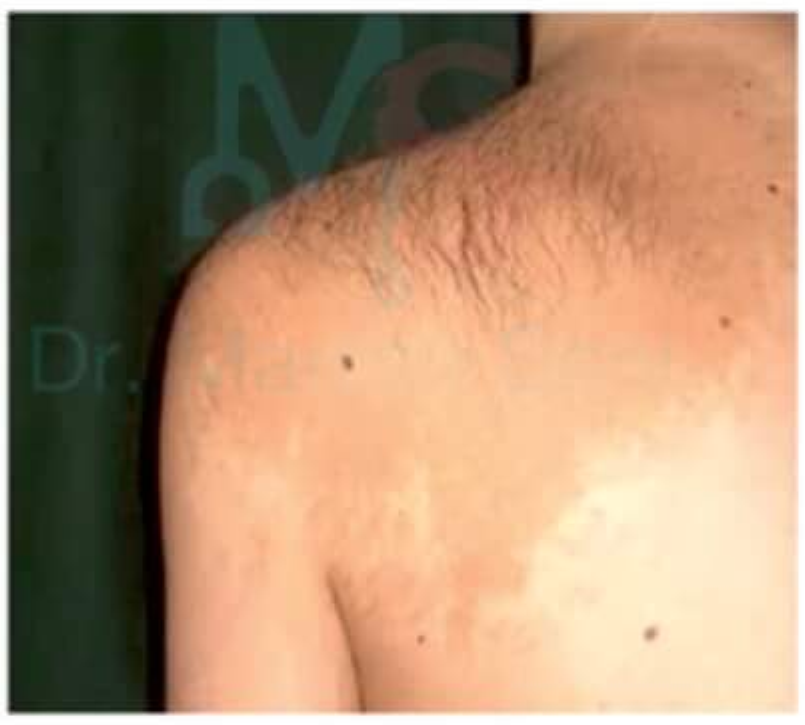

Focal vitiligo

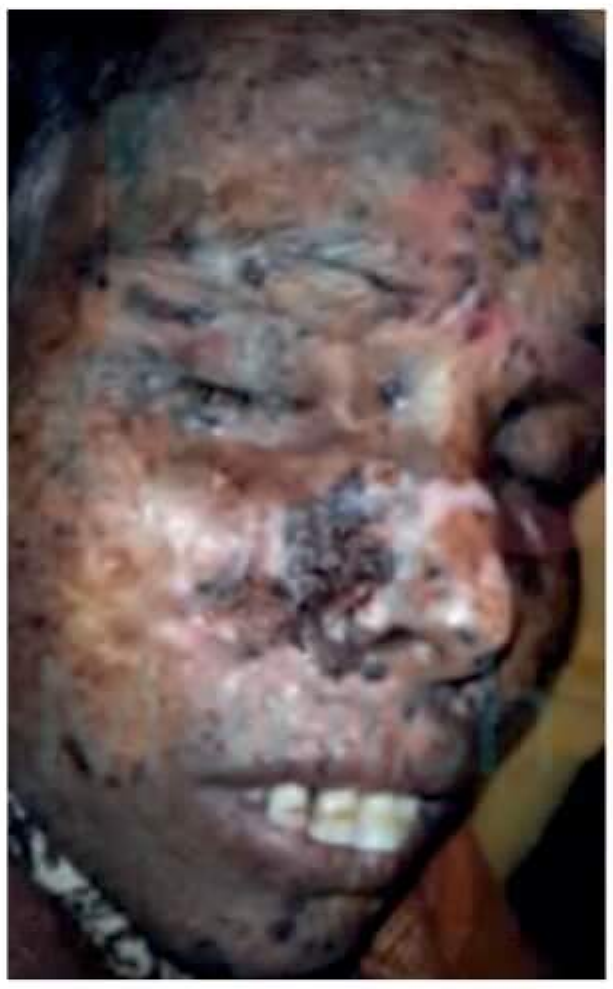

mucosal vitiligo

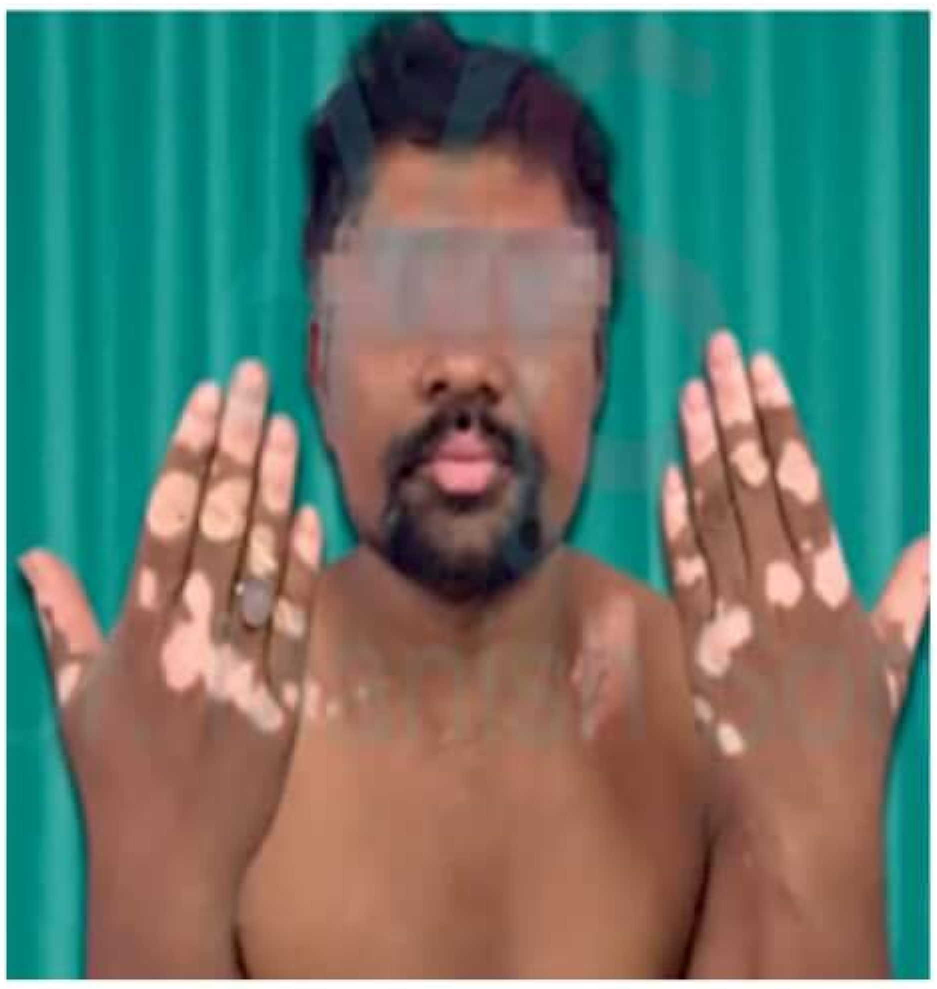

vitiligo with leukotrichia

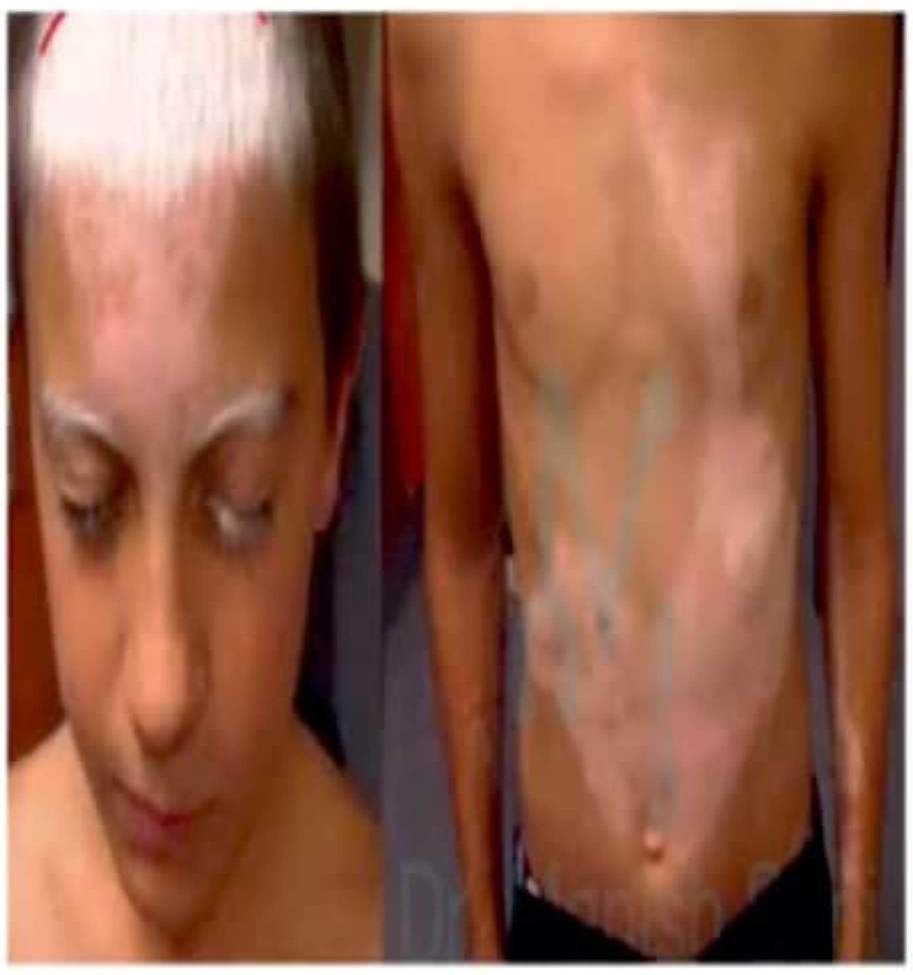

Trichrome vitiligo

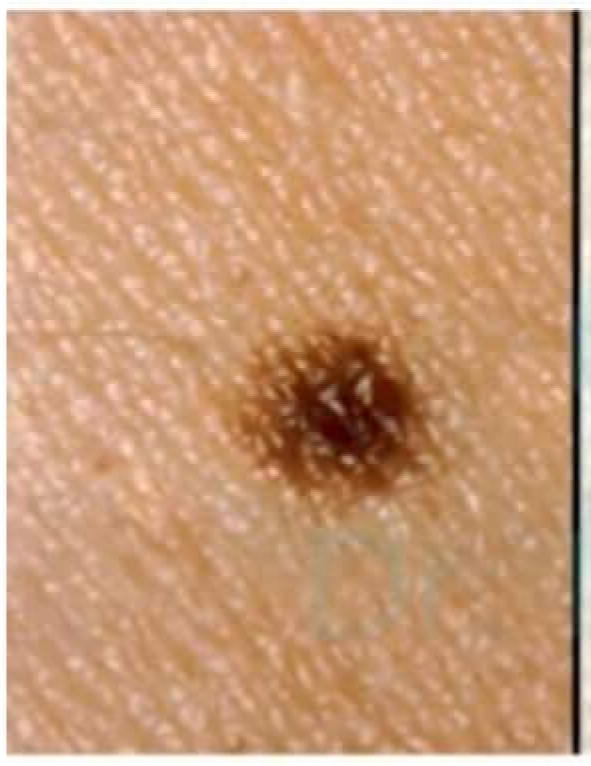

acanthosis nigricans

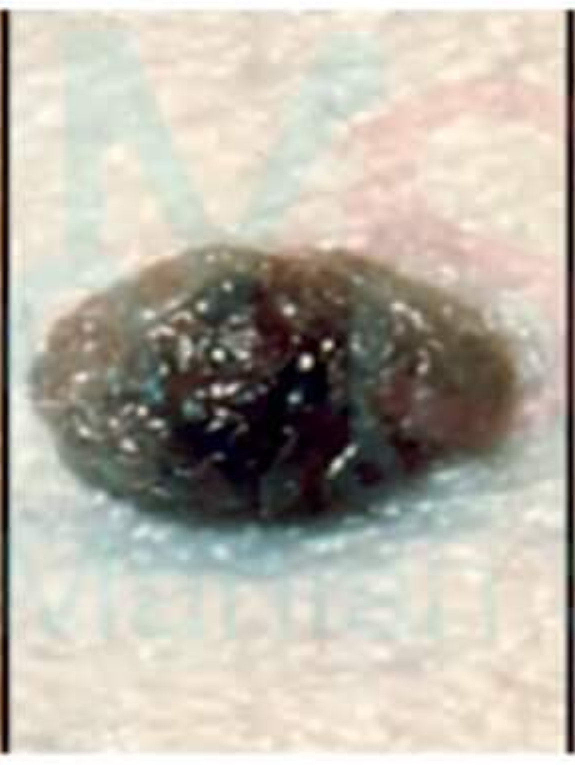

beckers nevus

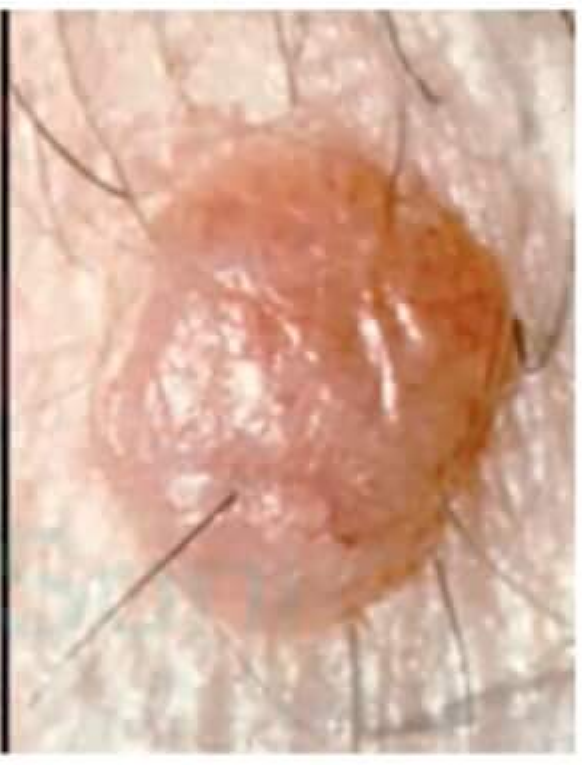

Xeroderma pigmentosa

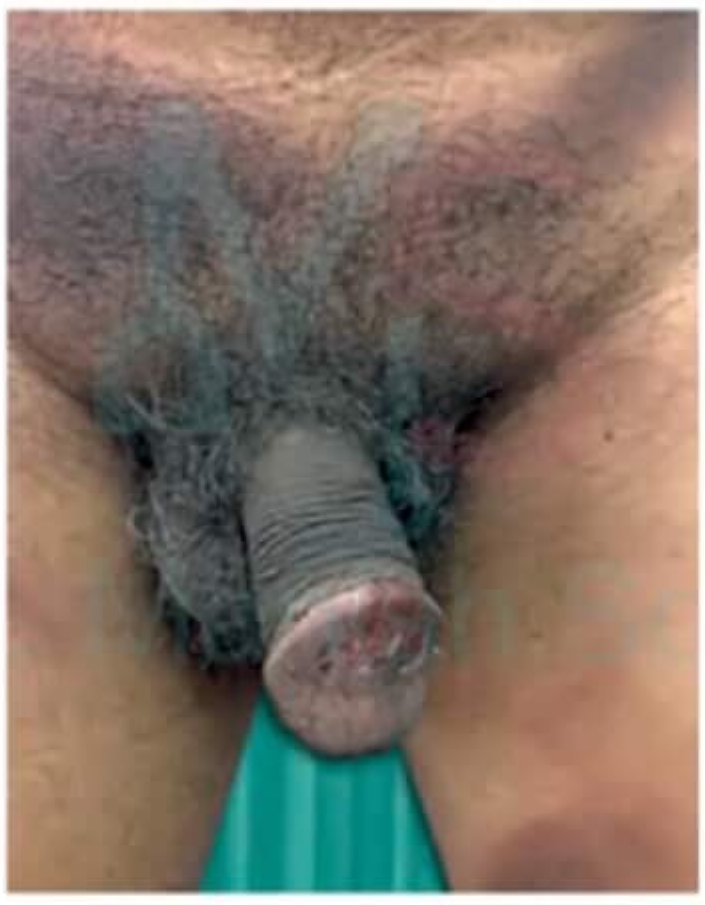

acrofacial vitiligo


piebaldism


JUNCTIONAL
MELANOCYTIC
NEVUS

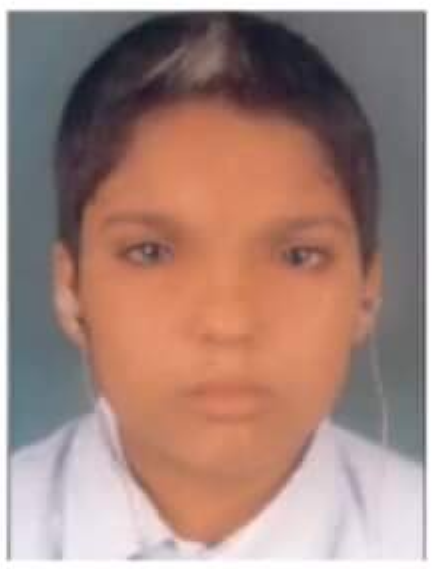

COMPOUND MELANOCYTIC
NEVUS


INTRADERMAL
MELANOCYTIC
NEVUS

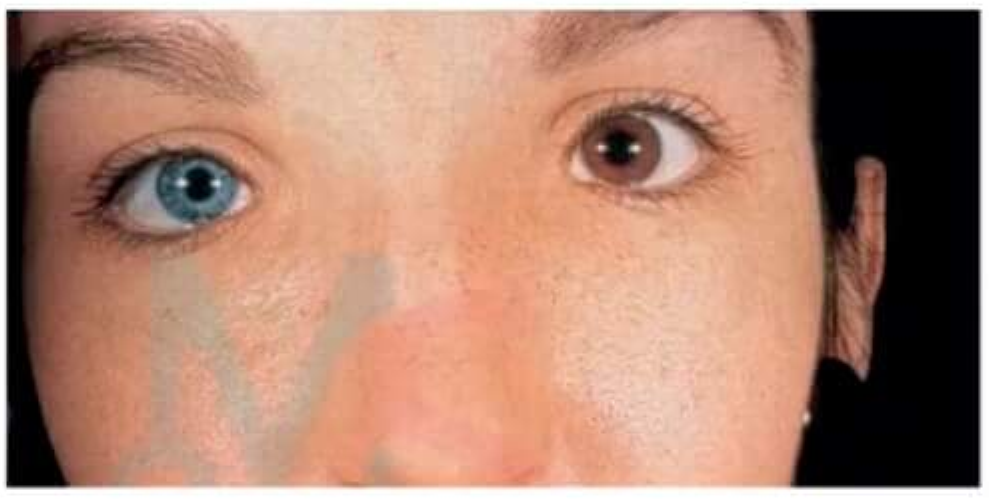


Fixed drug eruptions

UVA phototherapy device



white forelock, broad nasal root, dystopia canthorum, blue eyes in 10 years old indian girl with wardenburg syndrome

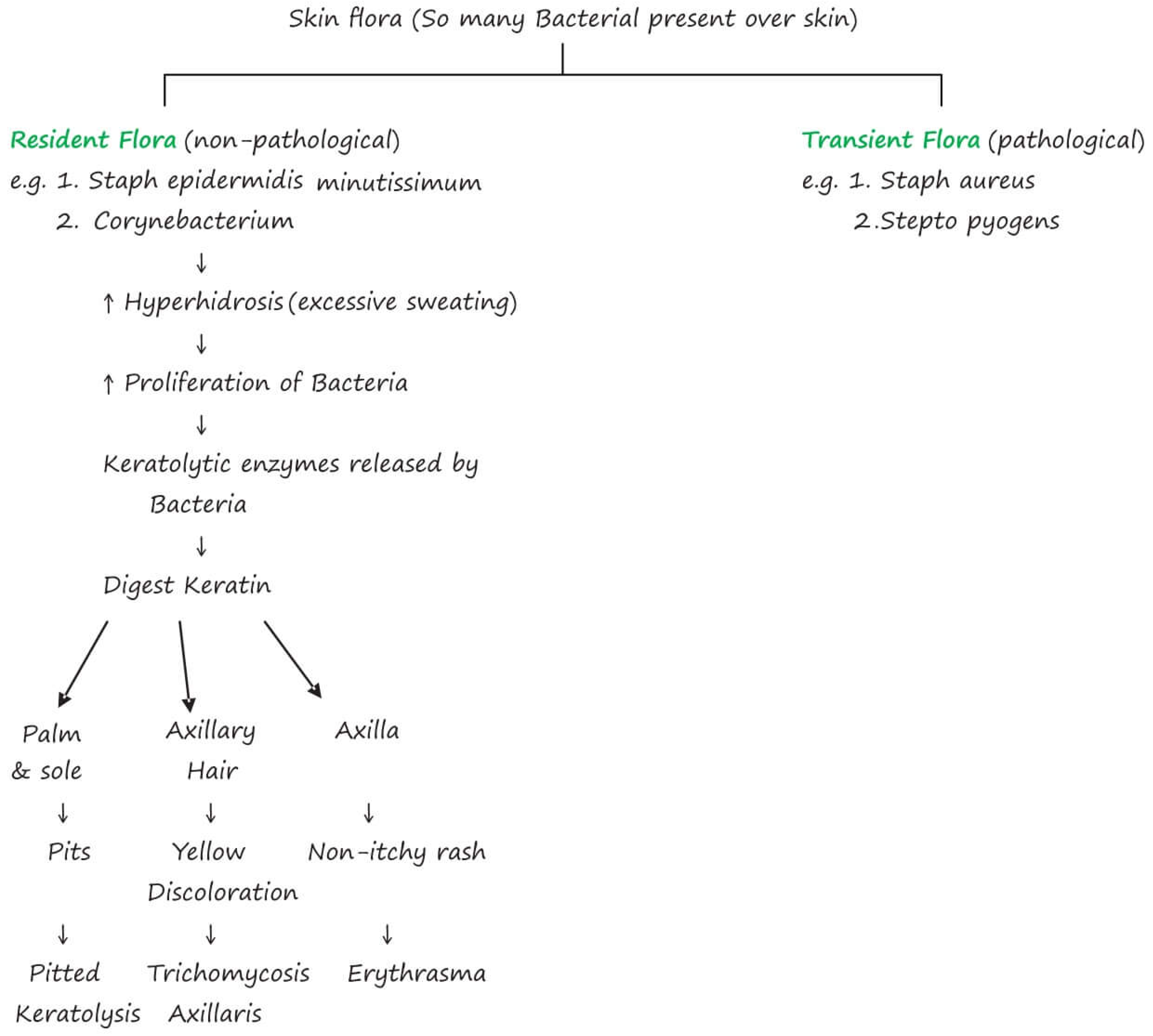


In the autosomal dominantly inherited disorder with variable expression, heterochromia of the iries, white forelock, SNHL deafness, broad nasal bridge, displacement of inner canthus and depigmented patches occur.

BACTERIAL INFECTIONS

Infections

1. **Bacterial Infection**


Skin flora (So many Bacterial present over skin)
Resident Flora (non-pathological)
e.g. 1. Staph epidermidis minutissimum
2. Corynebacterium
↑ Hyperhidrosis (excessive sweating)
↑ Proliferation of Bacteria
Keratolytic enzymes released by Bacteria
Digest Keratin
Palm & sole
Axillary Hair
Axilla
Pits
Yellow Discoloration
Non-itchy rash
Pitted Keratolysis
Trichomycosis Axillaris
Erythrasma
Transient Flora (pathological)
e.g. 1. Staph aureus
2.Stepto pyogens

Disorders caused by resident flora

1.Pitted Keratolysis:

→ patient shows hyperhidrosis which is a predisposing factor

→ characterized with multiple pits are seen on palm & soles

2. Trichomycosis Axillaris:

→ Here mycosis is a misnomer as it is a bacterial disorder caused by corynebacterium tenuis.

→ Clinically patient presents with yellowish discolorations over axillary hairs.

3. Erythrasma:

→ Caused by corynebacterium minutissimum

→ Clinically present with non-itchy rash over axilla (MC site) > groin

→ When examined under wood's lamp show coral red florescence which is due to coproporphyrin III

Rx for all above disorders : a. Topical: fusidic acid 2% (sodium fusidate)
b. systemic: clarithromycin 1gm stat. or erythromycin 250 QID 1wk

important facts

treatment for hyperhidrosis: Topical: →20% aluminium chlorhydrate
→ 1% formaline solution
Injection: → Botox
Others: → iontophoresis
Surgery: → sympathectomy

Disorders of transient flora:

1. impetigo

Classified into

	Impetigo Contagiosa (Most common) Non-Bullous	Bullous
1. **Causative organism**	Strep> Staph Aureus ↓ ↓ Mc in: developing developed nations	Staph aureus
2. **Clinical Feature:** -	Common in child Common site face Erosions- ^{Q} Golden yellow/ Honey colored crusting	→ Common in child → Common site face → Exfoliative toxins of staph destroy Dsg1 forming of subcorneal bullae → hypopyon sign i.e. pus is collected in lower half of bullae
3. **Complication:-**	Post Streptococcal Glomerular nephritis	Staphylococcal skin scalded syndrome features: -mucosal involvement is absent -Superficial (subcorneal epidermal Separation) -Nikolsky sign positive
4. **Treatment:-**	**Erythromycin**	**Dicloxacillin**

Cephalosporin
Topical Antibodies
→ Fusidic acid
→ Mupirocin
→ Retapamulin

NOTE: Condy's compression → is $KMnO_4$ solution 1:10,000 H_2O
→ it is antibacterial & help in removing crust

2. **Ecthyma** – it is a deeper variant of impetigo
   - → Clinically presents with indurated lesions with necrotic crust, which on removal leads to ulcer formation.
   - → Caused by:
     1. **Ecthyma pyogenicum**: – Strep, staph aureus
     2. **Ecthyma gangrenosum**: – Pseudomonas aeruginosa
     3. **Ecthyma contagiosum/**[Q]: – Parapox virus/ Orf virus
        Contagious pustular disease/Orf disease (it's a zoonotic viral condition spreads via sheep goat)

3. **Erysipelas**
   1. Causative organism: – Group A β-Hemolytic Streptococcus
   2. Borders: Sharp
   3. Toxicity Less ↓
   4. Associated with lymphangitis is more common.

4. **Cellulitis**
   1. Both (Streptococcus & staph aureus)
   2. borders: Diffuse
   3. more toxic ↑
   4. association with lymphangitis is less common

Important Fact

1. **Milian Ear sign** → cellulitis involves deeper subcutaneous tissue so ear involvement is absent (as its devoid of subcutaneous tissue) whereas in erysipelas ear involvement is seen. Which helps differentiating facial lesions only.
2. **Erysipeloid** → caused by Erysipilothrix rhusiopathea
3. **Wood lamp**:- 320-400 nm light is used 365 nm peak
   → Filter – Barium silicate & 9% Nickle oxide

Disorder	Color
1. Tinea Capitis	Green
2. Pseudomonas	Green
3. Porphyria(urine)	Pink
4. Squamous Cell Carcinoma	Red
5. Erythrasma	Coral Red
6. Pityriasis versicolor	Yellow/Apple green
7. Vitiligo	White/ivory white
8. Ash leaf macules (Tuberous Sclerosis)	Bluish white
9. tetracycline stained teeth	yellow/orange
10. chromhydrosis (can be due to lipofuscin, drug: mepacrine, dyes)	variable

4. - **Ritters disease** aka SSSS/ pemphigus neonatorum
   - **Reiters disease** aka reactive arthritis

     Clinical features of reactive arthritis:

     C- conjunctivitis
     U- urethritis
     B- balanitis (circinata)
     I- iritis
     K- keratoderma blennorrhagica
     A- Arthritis

5. **Dermatitis artefacta**: artificial dermatitis i.e. self-inflicted injuries seen in psychiatric or hysteric patient or sometimes in prisoners also, which don't fit into any disease pattern.

Q. 28 years old females presented with shallow discreate and coalescent pits, hyperhidrosis, and mal odour over her palm & sole. Diagnosis?

A. Tinea pedis
B. Palmoplantar keratoderma
C. Pitted keratolysis
D. Inverse tinea versicolor

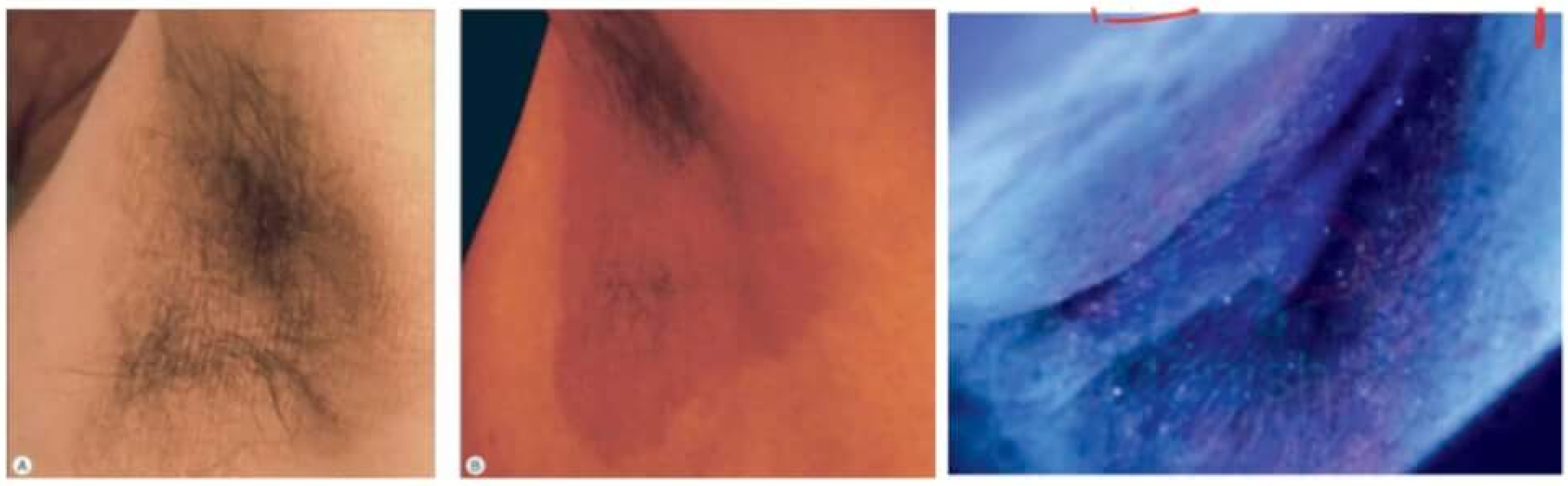
Ans- C

Q. Following instruments is commonly used to diagnose all the following conditions except?

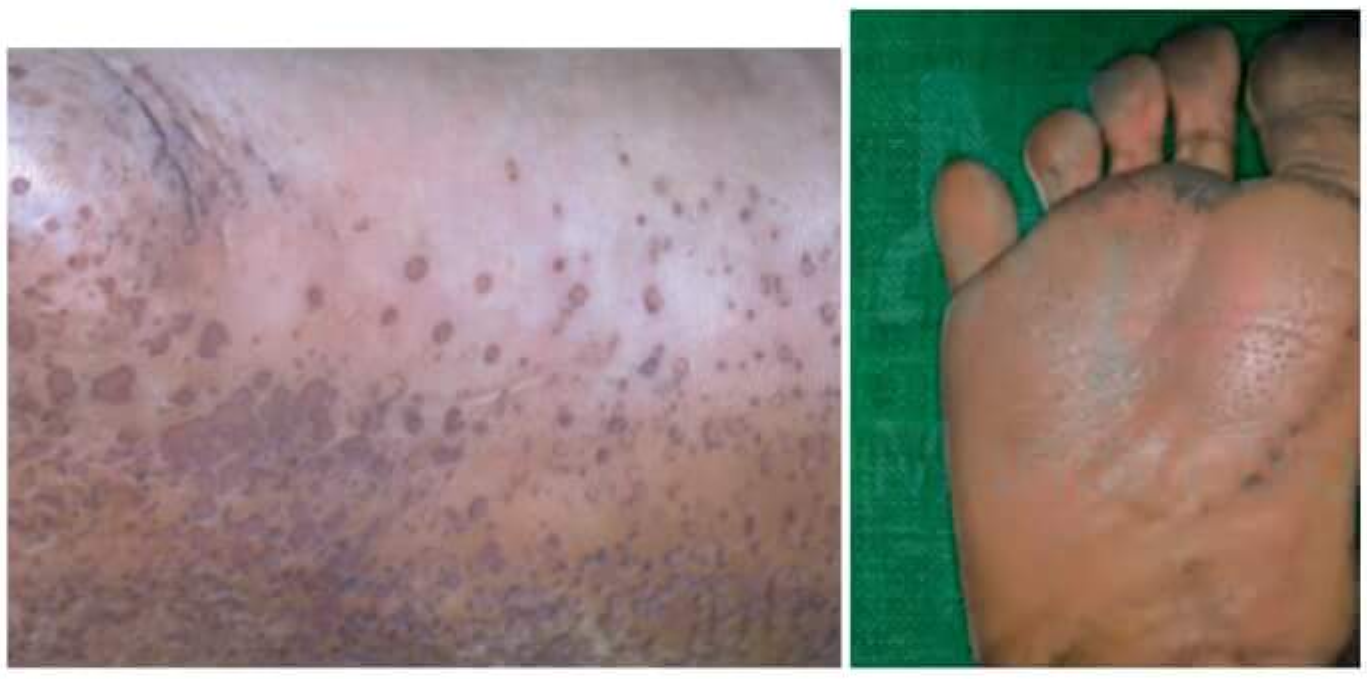
A. Staining of teeth or sebum from tetracycline
B. In investigation of dermatitis artefacta
C. Squamous cell carcinoma
D. Bromhydrosis

Ans- D

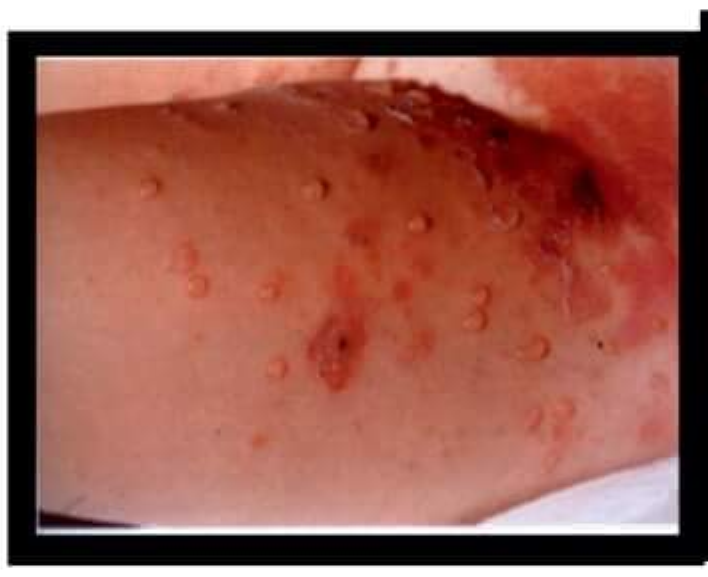
Wood's lamp



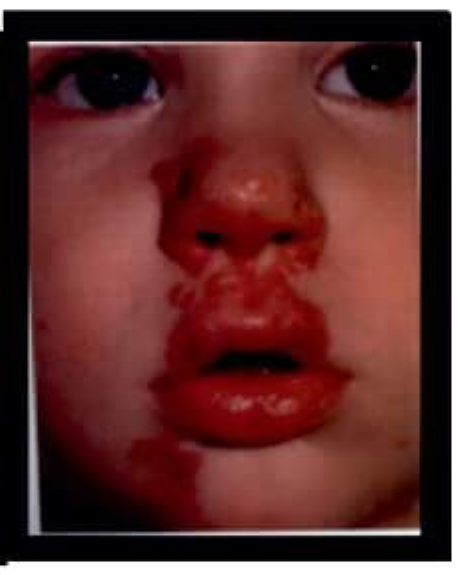
Erythrasma axilla coral red on woods lamp



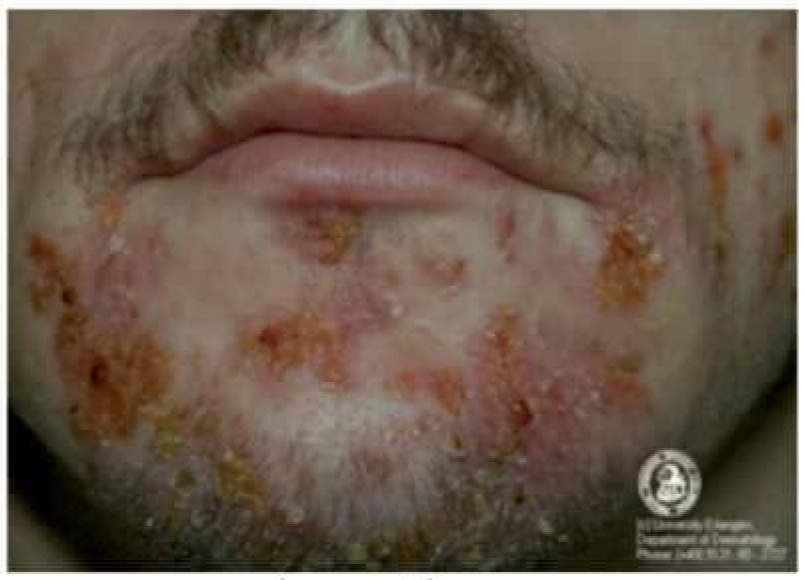
pitted keratolysis



Bullous impetigo

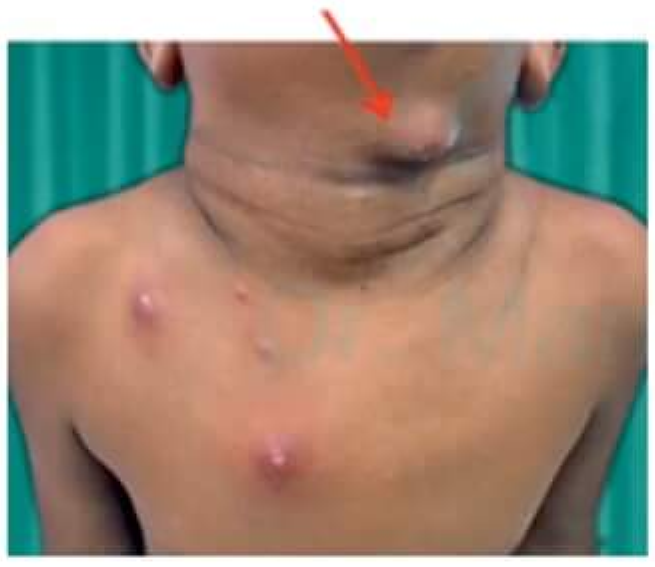


impetigo contagiosa

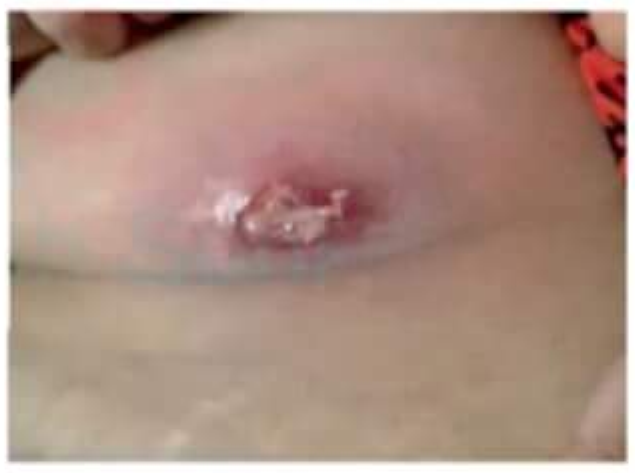


impetigo

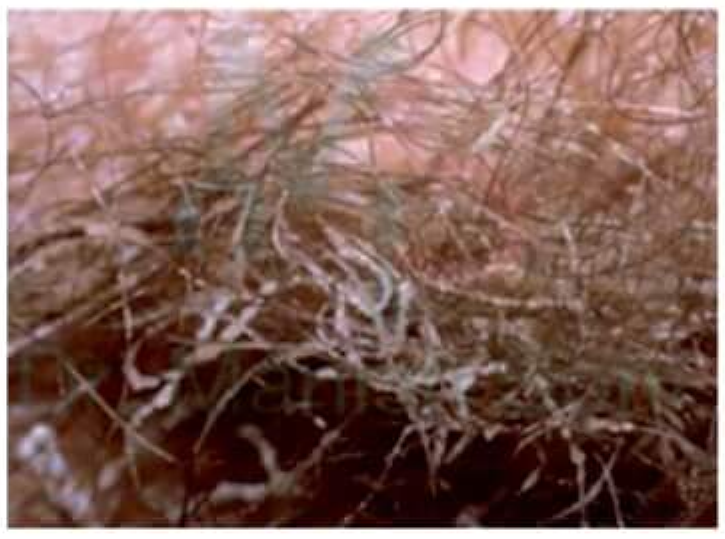
furuncle (boil): deep seated inflammatory nodule around hair follicle

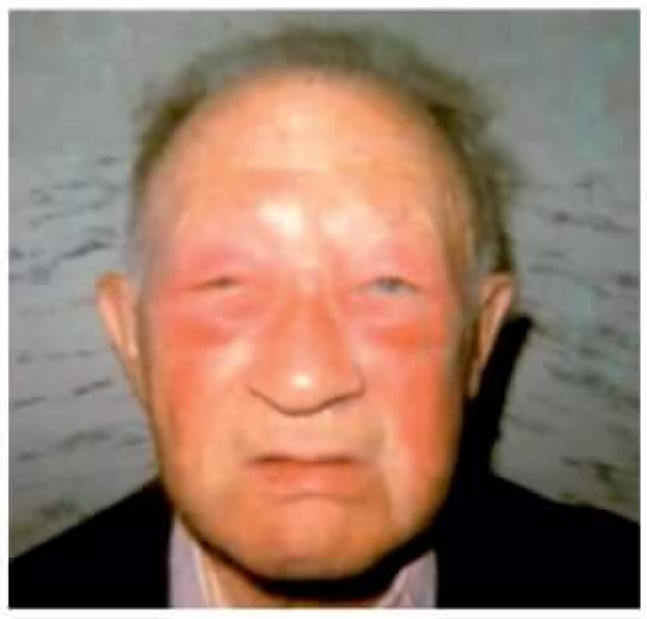


carbuncle: extensive, deeper infection of contiguous hair follicles, common in dibetes and patients on steroid therapy.

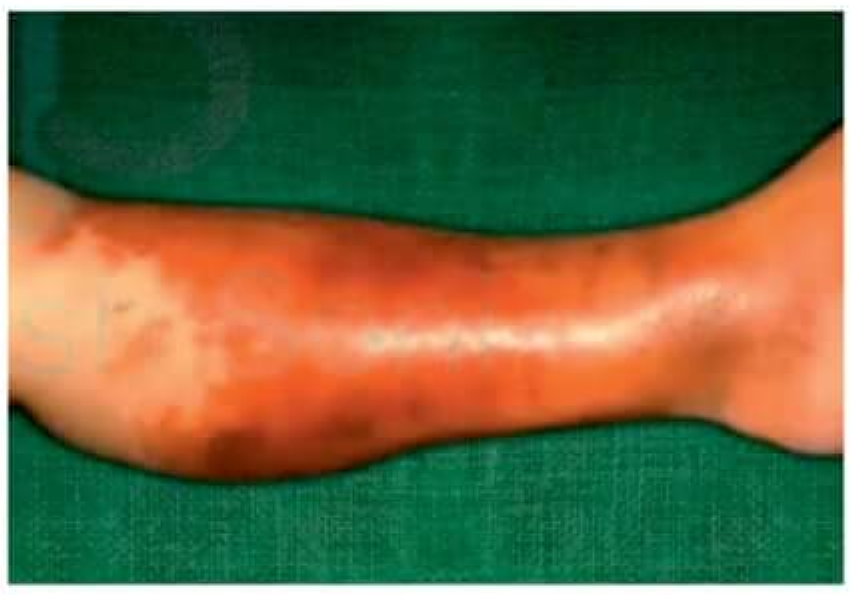


lesions discharging pus from sieve like openings seen

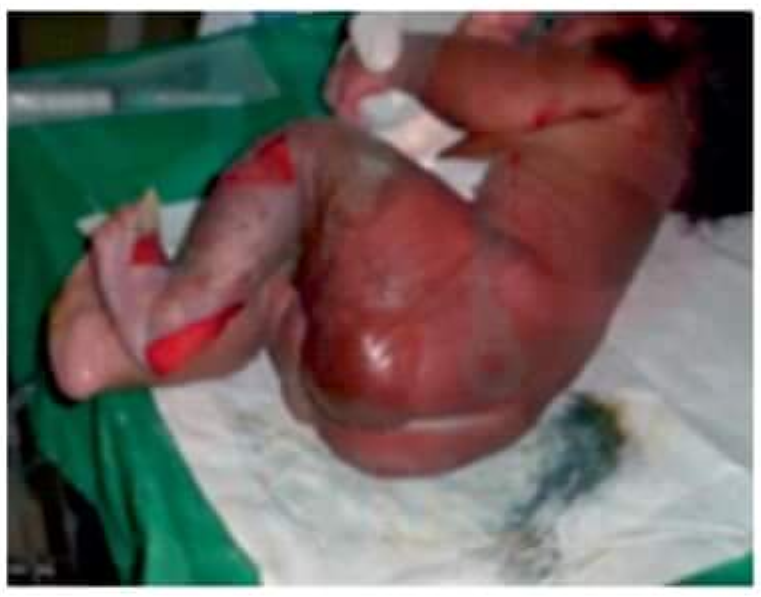




Erysipelas



Cellulitis



SSSS with positive nikolsky sign

FUNGAL INFECTION

Fungal infection

↓ Superficial ↓ Deep

I. Superficial fungal infection

1. Pityriasis Versicolor

↓ Scaling ↓ Various

↓

Lesions are hypopigmented > Hyperpigmented

- → **Causative agent**: Malassezia globosa > Malassezia furfur
- → **Clinical feature:**
  - a. Peri-follicular[Q]
  - b. Hypopigmented macules
  - c. (Small macules combine to form) Patches
  - d. Rice powder like (furfuraceous) scaling is seen - Indicates nail scratch sign positive
  - e. **Common site of lesion**: upper trunk

Inverse pityriasis versicolor: This Diagnosis is formed if lesions present on Extremities and trunk is spared

→ Lesion can be hypopigmented / hyperpigmented

↓ (hypopigmented):

- Due to Azelaic acid[Q] (tyrosine kinase inhibitor)
- ∴ No melanin
- ∴ Lesion are hypopigmented
- Secreted by Malassezia

↓ (hyperpigmented): due to increase in size of melanosomes

→ Investigation 10% KOH mount shows:

Short fungal hyphae with fungal Spores

↓ ↓

giving spaghetti & meat ball appearance

Banana & Grapes appearance

→ On wood lamp Ex: yellow / Apple green

→ Treatment: Azoles

Topical: Ketoconazole

Systemic: fluconazole

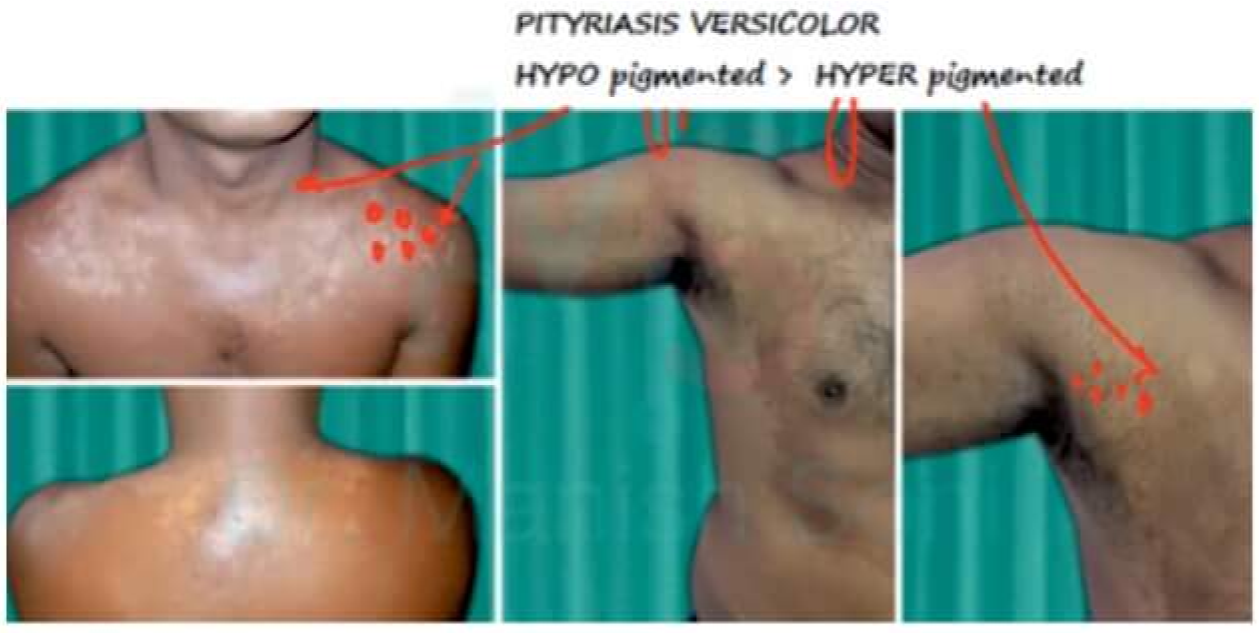
Q. which antifungal not effective in pityriasis versicolor?

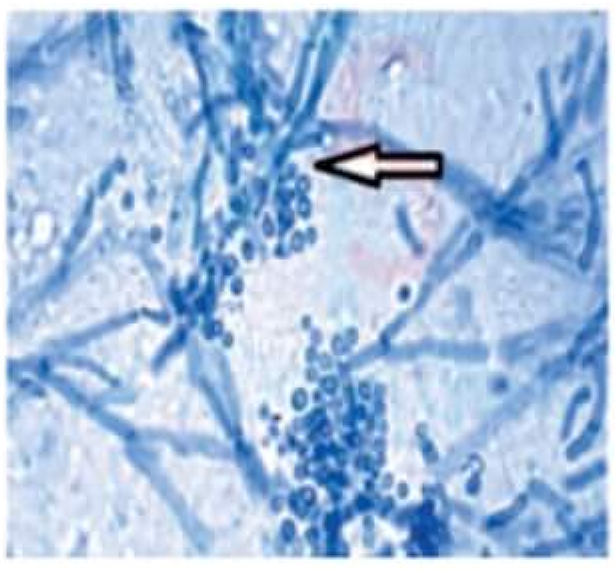
A. Griseofulvin

Old Rx used: white filed ointment ie 3% salicylic acid + 6% Benzoic acid

MOA was: It helped in skin resurfacing, acid penetrating into superficial layer & slough off, which is replaced by normal skin layer below it

Not used anymore as acid has side-effects like burning, irritation, pigmentation





Spaghetti and meat ball
Appearance on 10% KOH

2. Tinea Nigra.

- Causative agent: pheoanellomyes werneckii
- Lesion are black patch over palms which respond well to topical antifungal like ketoconazole.
- it is common in western countries in fairer skin population – {not so common in India}

3. Candidiasis:

Causative agent: → Candida albicans
→ Candida gabrata

Depending on site are various type :

i. Oral candidiasis: oral Mucosa, Buccal mucosa

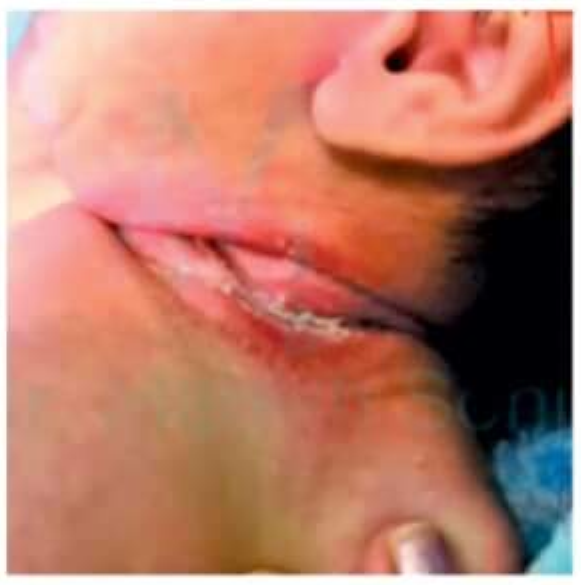
a. Acute pseudomembranous candidiasis: → MC type (Aka thrush)
↓
As it can be detached

b. Acute Atrophic candidiasis:
c. Chronic atrophic candidiasis
} Both present with red color lesions

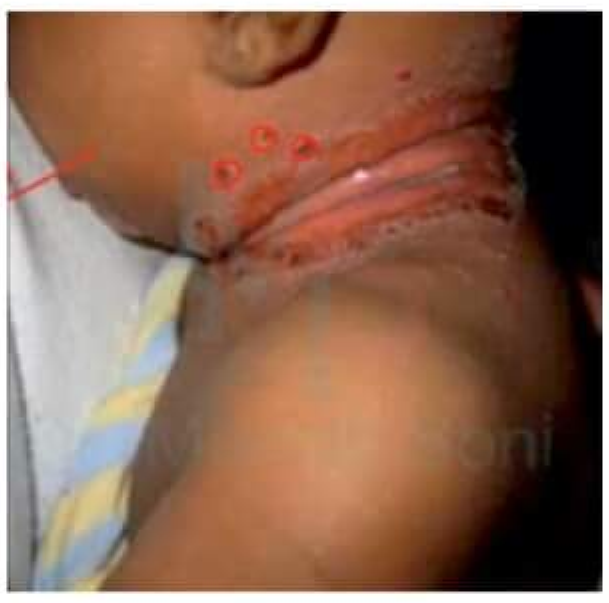
Common in old age esp people who wear denture: Aka denture candidiasis

d. Chronic hyperplastic candidiasis → Common in smokers.

e. Perleach: Candida infection of angle of month



Candida intertriginous



satellite lesions

ii. Vaginal candidiasis:

- Clinically present with thick curdy vaginal discharge

Vaginal discharge

Candidiasis (C=C)
White creamy curdy
Discharge with Itching

(TGI Friday)Trichomoniasis
greenish frothy discharge
associated with severe itching

Bacterial
(WHIFF THE CLUE OF FOUL PLAY)

- scanty white
- Adherent discharge
- foul smelling
- clue cells present
- whiff test positive

RX: Fluconazole 150mg stat

treatment
Secnidazole
2gm stat

NACO kit-2 {Green color}

Used for Rx of vaginal discharge {syndrome management where better lab facility not available

iii. Candidal Balanoposthitis

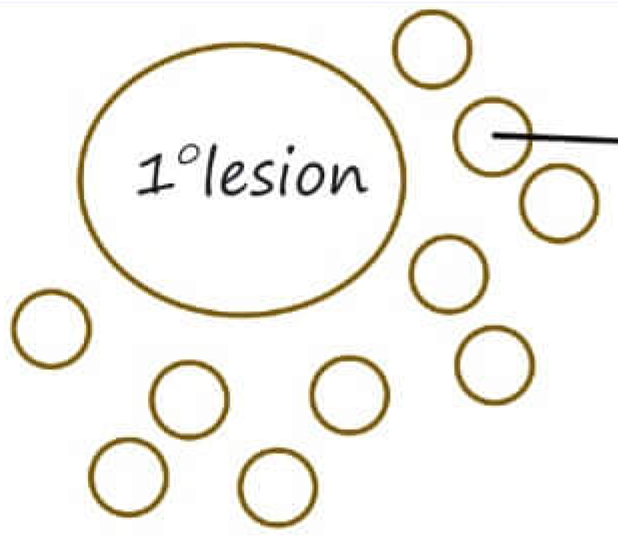
- It is recurrent in Diabetes mellitus patient
- Present with fissures over prepuce

iv. Candidal paronychia

Candidial infection of nails folds

v. Candidal intertrigo – infection intertriginous areas {I.e flexor site, groin, axilla, infra mamillary folds.

- Clinical feature present with satellite lesions i.e. lesions around 1° lesions, they are vesico-pustular lesions.



Satellite lesions
↓ seen in

mnemonic "BSC"

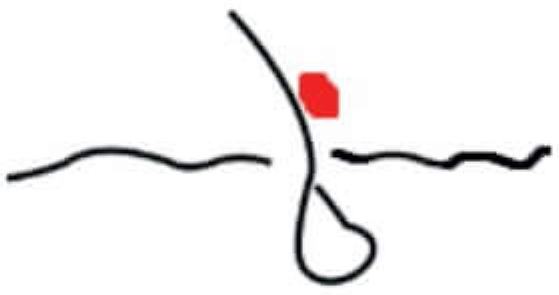
1. Borderline Tuberculid type of Hansen's disease(Leprosy)
2. Sporotrichosis
3. Candidiasis

- Investigation: 10% KOH → shows pseudo hyphae
- Rx: Drug of choice fluconazole
- Griseofulvin not effective.

Pseudo diaper rashes: candidal infection seen in diaper area in infants

Erythema of Jacquet: are chronic, persistent candidal erosions
Etiology: multifactorial – occurs at site: Excessive sweating skin maceration

4. Trichomycosis nodularis / Piedra- it is fungal nodule attached to hair follicle



	2 types	
	Black aka dark Piedra	**White aka light Piedra**
C. Ag	Piedrae hortae	Trichosporon beigelii
Hair of:	Scalp	Axillary public
Nodules	Firmly attached	Loosely attached +

treatment

Shaving of hair
Topical of antifungal

Shaving of hair:
- Required as infection is not only on surface But beneath it
- to avoid recurrent infection cut hair in that area
- If generalized: shave off

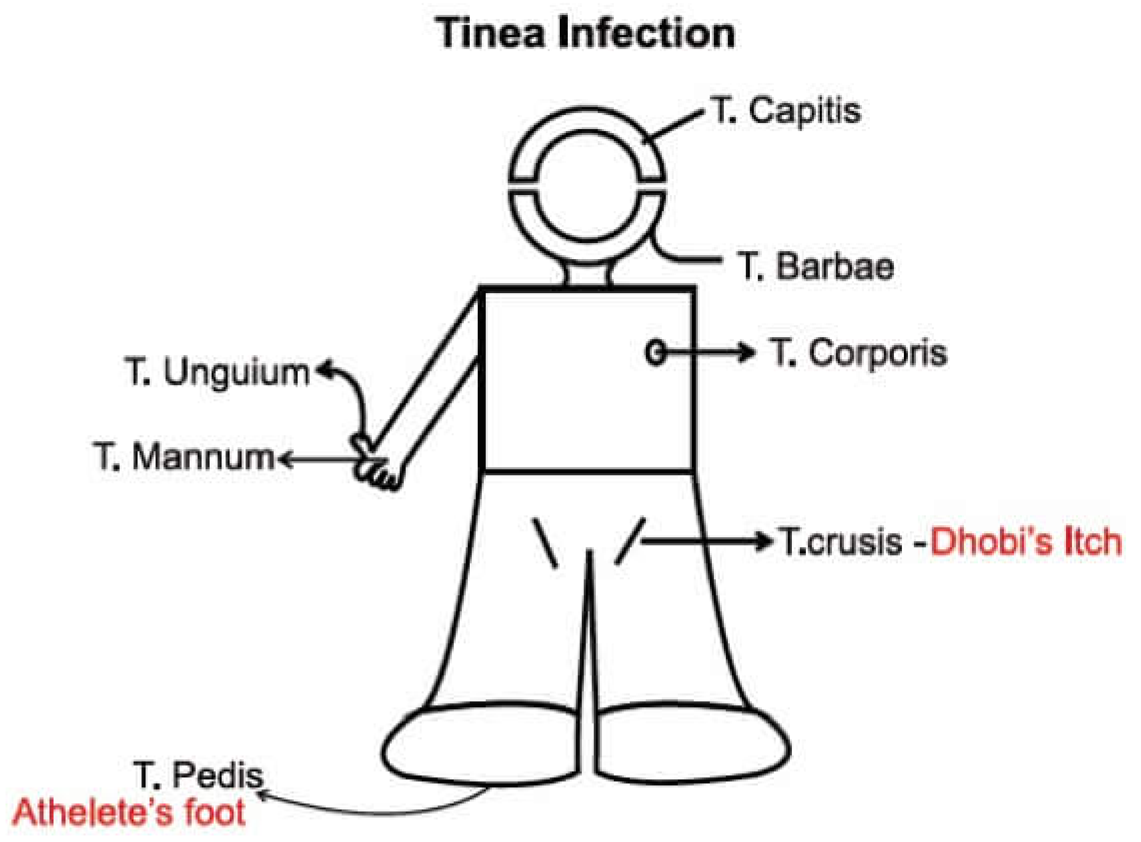
Topical of antifungal: for females where shaving can't be done (but If localized: recurrence is possible)

4. Dermatophytosis:

Fungal infection caused by dermatophytes

3 genera	**Skin**	**Hair**	**Nail**
1. Trichophyton	✓	✓	✓
2. Epidermophyton	✓	x	✓
3. Microsporum	✓	✓	x

Dermatophytes cause tinea infection Depending on site various types



i. Tinea corporis/ T. glabrosa / T. circinate

- Annular lesions, active margins (ring like)
- Shows Central clearing

> - Central crusting → leishmaniasis
> - Central scarring → lupus vulgaris

ii. Tinea imbricata.

- Causative agent: Trichophyton concentricum
- Shows multiple concentric active margins

iii. Tinea incognito.

- Unrecognizable
- Under effect of steroids inflammation and symptoms subside BUT infection persists

iv. Tinea manuum.

Generally unilateral present with hyperlinear & hyperkeratotic lesions

v. Tinea pedis

a. Interdigital (mc) → (Mc site 4th web space)
b. Squamous
c. Bullous
d. Ulcerative (rarest)

vi. Tinea capitis

2 types Broadly

- Inflammatory
  - Kerion
  - Favus
- Non inflammatory
  - Black dot
  - Grey path

Kerion:

- Commonly seen in children
- Shows boggy tender swelling
- Easily pluckable hair
- Associated with regional lymphadenopathy
- Causative agent: Trichophyton mentagrophyte (jipmer 17)
  Trichophyton verrucosum

Favus

- Commonly seen in residents of Kashmir
- Foul smelling thick crust k/a scutula seen over scalp
- Causative agent: trichophyton schoenlenii (MC)

Black dot

- Caused by endothrix species
- On pulling hair break at level of scalp
- On patch examination (under microscope) shows black dot
- Common org. Trichophyton tonsurans
  Trichophyton Violaceum

Grey patch:

- Caused by ectothrix species
- On patch examination: broken hair follicles & greyish patch
- Hair break above the level of scalp
- common org. Microsporum canis

- Wood's lamp Examination: → green florescence
  [Microsporum species and trichophyton Schoenlein are fluorescent]
- Drug of choice – griseofulvin 10-15 mg / kg / bwt

Important fact

1\.

Causative agent	DOC
• Trichophyton	Terbinafine
• Microsporum	Griseofulvin > itraconazole

2. MC ectothrix species causing tinea capitis: M. canis C
Mc endothrix species causing t capitis: T. Tonsurans T
2nd mc endothrix species causing t capitis: T. Violaceum ↓ V
Mc species causing Tinea capitis in india: T. Violaceum ↑ V
Mc species causing Tinea capitis in western: T. Tonsurans T
Mc species causing Tinea capitis in world: M. canis C

MC species causing: T. cruris
T. Corporis
T. Manuum
T. Pedis
T. Unguium
} Trichophyton rubrum DOC: Terbinafine

vii. Tinea unguium (onychomycosis)

↓ Dermatophytic fungal Infection of nail
↓ Fungal infection of nail

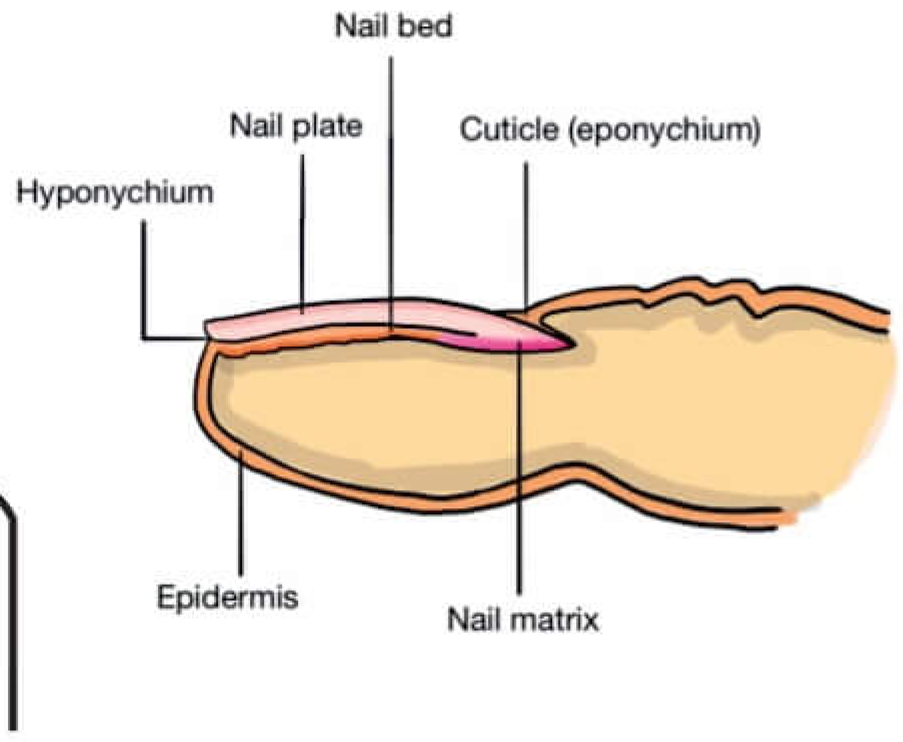
It is of 3 types

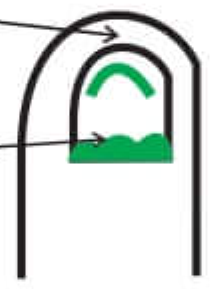
a. Distal lateral subungual onychomycosis (DLSO) MC

b. Proximal subungual onychomycosis (PSO) Rarest seen in HIV positive

c. Superficial white onychomycosis (SWO)

All these 3 types may lead to total dystrophic onychomycosis (TDO)





DOC – [Terbinafine (systemic)] > itraconazole
↓
May '18 AIIMS

> Q. Facing resistance to which of the following antifungal in last 2 years?
> A. Terbinafine.

Since longer duration therapy required give pulse therapy (consists of gap of 3 weeks)

i.e 250mg BD terbinafine X 7 days → Gap of 3 weeks ← 200mg BD terbinafine X 7 days

Gap of 3 weeks
↓
Continue for at least 3 to 4 cycles

Topical agents

- → Amorolfine
- → Ciclopirox olamine

} Available in form of nail lacquer (Weekly application)

II DEEP FUNGAL INFRACTIONS

1. Sporotrichosis/ rose gardener's disease/ rose thorn disease

- → Common in rose gardeners, florists
- → Causative agent – sporothrix schenckii
- → infection at site of prick

3 Variants	Clinical feature	Drug of choice
Cutaneous	Nodule with ulceration	Itraconazole
Lymphatic (MC)	Linear nodule with satellite lesions	Saturated solution of KI (oral) ^ itraconazole ,used to avoid s/e related to thyroid
Systemic	Upper lobe of lung involvement	Amphotericin B

- → Histopathology → Asteroid bodies

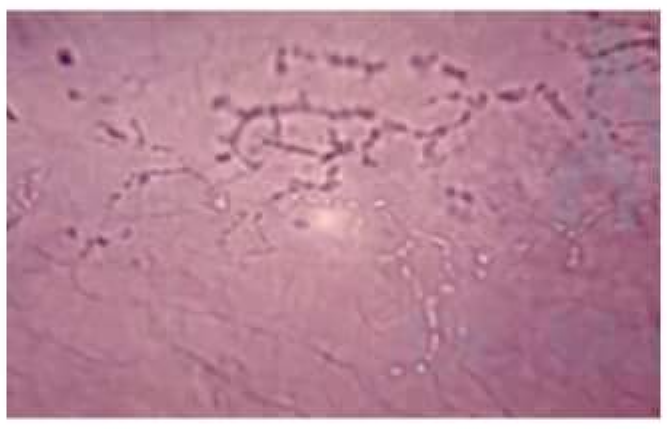
2. Chromoblastomycosis / fonsecaea disease

↓

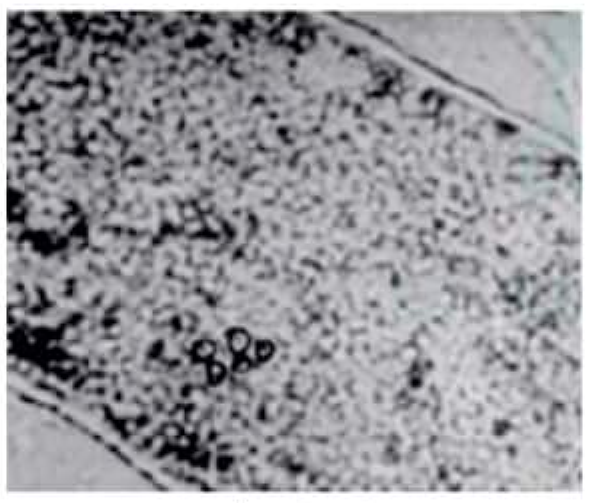
Pigmented / colored

- **Fungal infection by pigmented fungus**
    1. Fonsecaea compacta
    2. Fonsecaea pedrosoi
    3. Pheallophora verrucosa
- **Clinical feature:** Rough keratotic verrucous/ warty lesions which may later form giant plaque cauliflower like growth
- **Histology:** Medlar/ Sclerotic/ Copper penny bodies

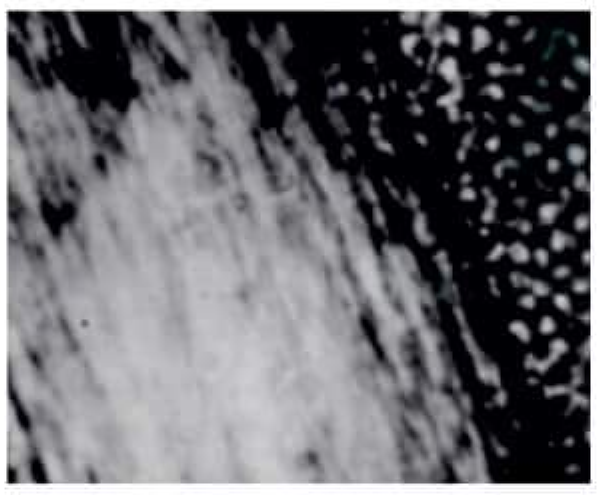
DOC: Itraconazole



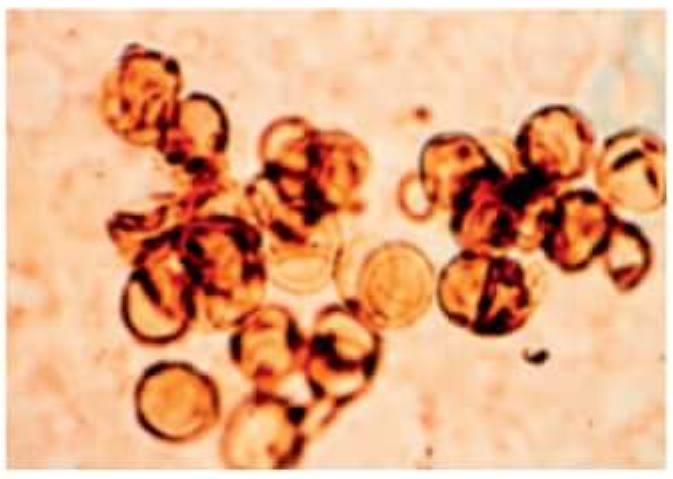
KOH mount showing septate hyphae(mycelia)



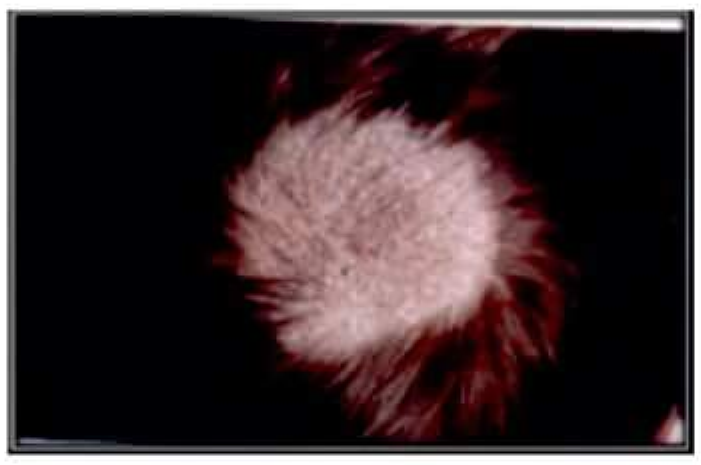
Endothrix
(spores are inside hair follicle)



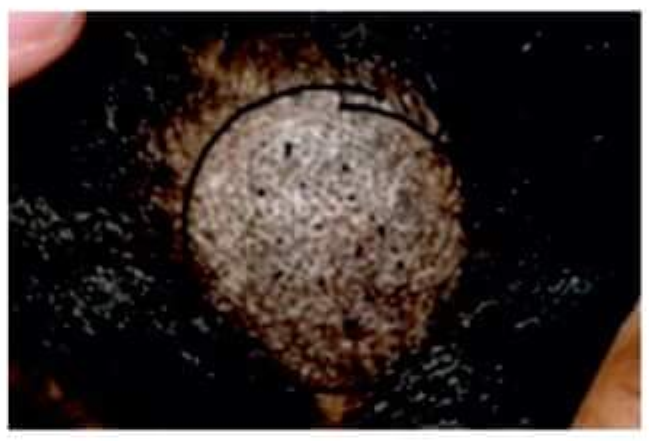
ectothrix
(spores are outside)



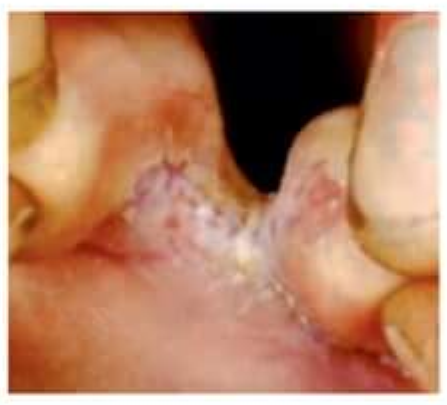
Copper penny bodies
Thick walled structures 4-12 um in diameter



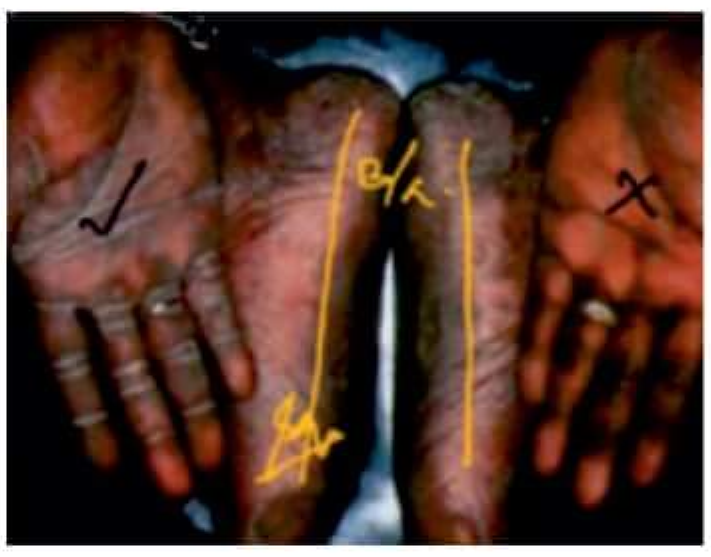
kerion-tinea capitis
(inflammatory)

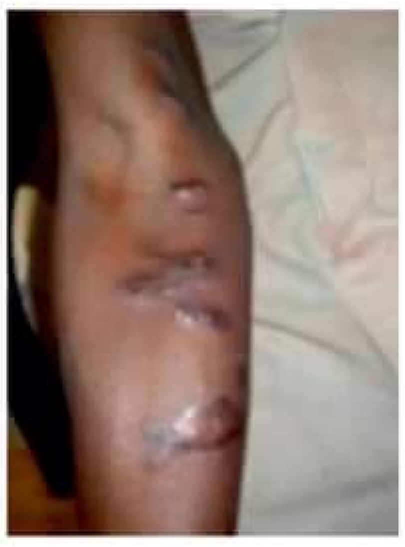


Black dot- tinea capitis
(non inflammatory)

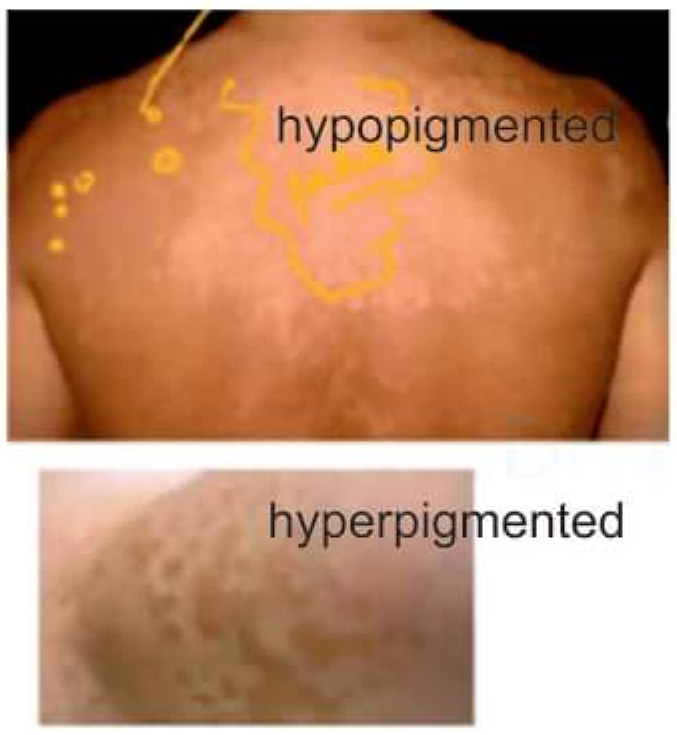


tinea pedis

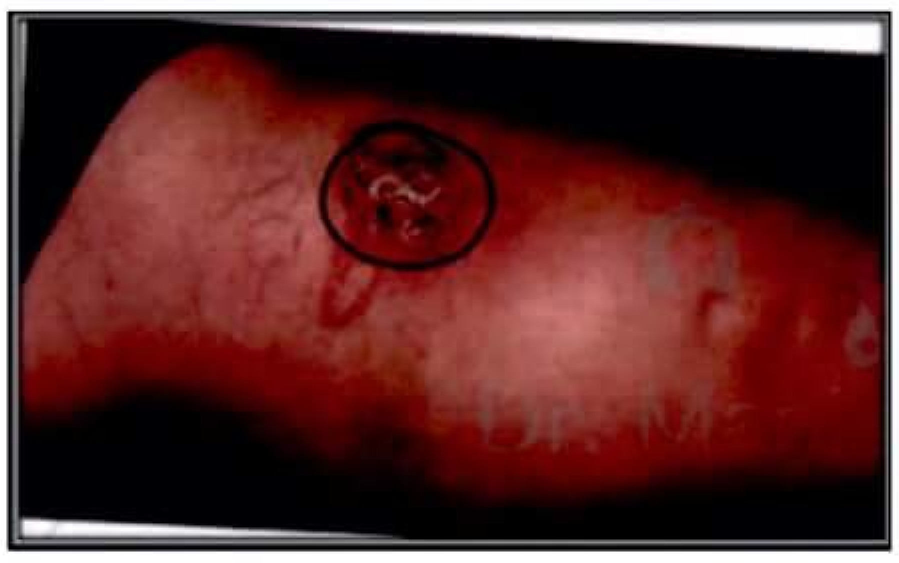




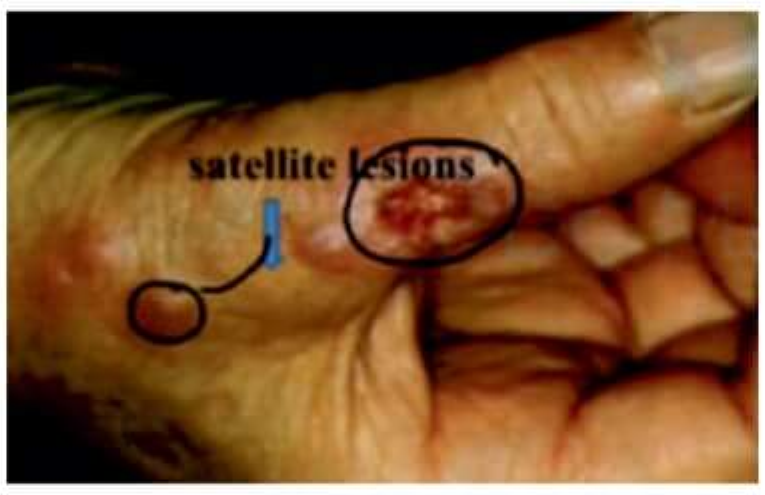
One hand and two feet syndrome sporotrichosis
(nodule developing from draining lymphatics)



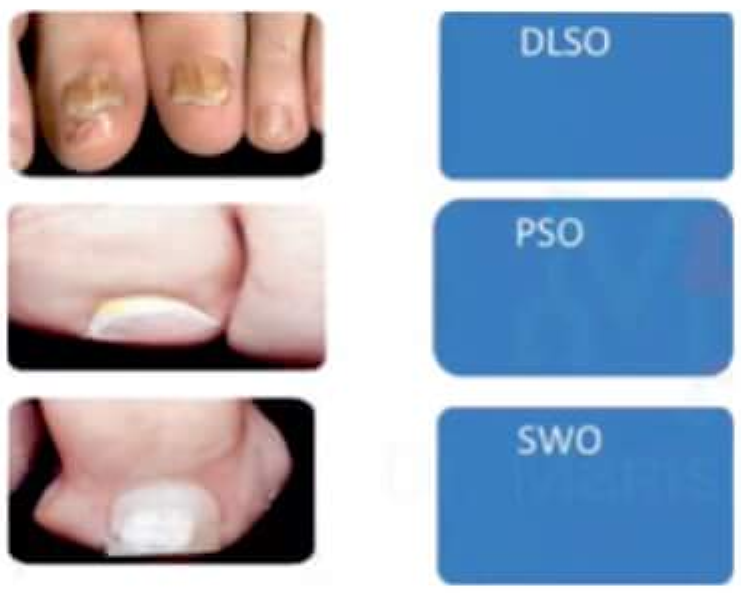
Pityriasis versicolor



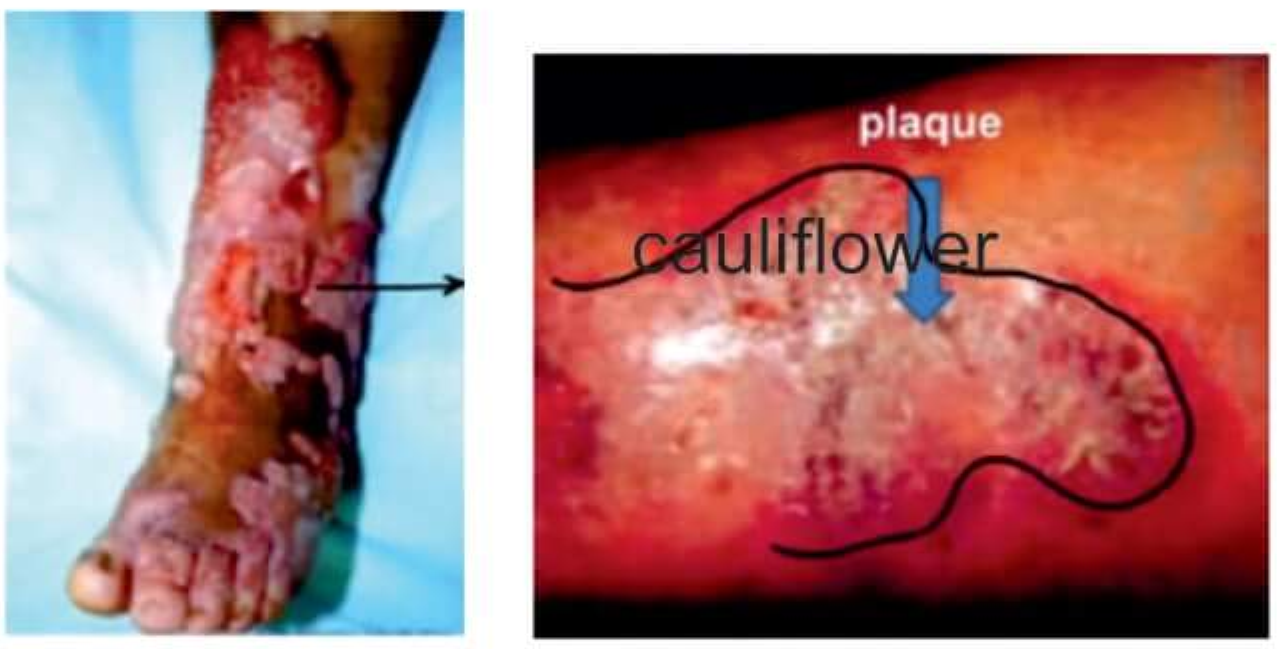
chromomycosis



Ulcerated nodule reveals the satellite lesions characteristic of lymphangitic



tinea pedis



chromoblastomycosis

MYCOBACTERIAL INFECTIONS

- Cutaneous tuberculosis
- Leprosy

I. Cutaneous Tuberculosis: -

- **Exogenous**
  - Eg.i) TB Chancre
  - ii) TBVC/Anatomist's wart
- **Endogenous**
  - i) Lupus vulgaris
  - ii) Scrofuloderma
  - iii) Orificial TB
- **Tuberculids**
  - -due to mycobacterial hypersensitivity

→ Most common type of cutaneous TB is Lupus vulgaris

→ M/C type in children is Scrofuloderma

→ Most cases of lungs vulgaris are due to hematogenous spread.

→ Contiguous spread seen in Scrofuloderma

→ Orificial Tb occur due to autoinoculation.

Exogenous

i) **Tuberculous Chancre:** Primary Inoculation TB

- Rare
- Presents with ulcer with undermined edges

Undermined edges also seen in

→ Chancroid

→ Pyoderma gangrenosum

ii) **Tuberculosis verrucose cutis (TBVC)/ Anatomist's wart**

→ post primary inoculation TB.

→ Seen in people with good cell mediated immunity (good resistance)

→ Patient present with warty, solitary growth over exposed area. (feet, hand)

→ Lesions are verrucous (warty)

→ Seen in anatomy professors and pathologists.

Viral warts	Anatomist warts
1.Numbers: multiple	Single
2.Induration: Absent	Present
3.Scarring: Absent	Present(central)
4.Discharge: Absent	Present(Serous)

Endogenous

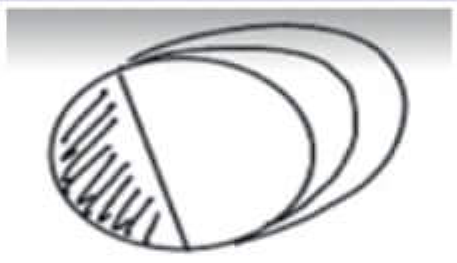
i) **Lupus Vulgaris** (Most common type)

→ Erythematous/violaceous plaque with central scarring ^{Q}

→ On diascopy ^{Q}

Apple jelly nodules are seen at the periphery of lesion^{Q}

Central Clearing seen in :Tinea[Q]
Central crusting seen in: Leishmaniasis[Q]
Central Scarring seen in: Lupus vulgaris[Q]



Lesion extend from one side and leave scarring on another side

i) **Scrofuloderma**

→ Most common in children
→ Cutaneous involvement secondary to underlying Tubercular focus (Lymph Node, Bone, joint)
→ Presents with multiple sinus with discharge
→ Most common Lymph node involved: Cervical

ii) **Orificial TB**

→ Auto inoculation
→ Perioral lesions present

Tuberculids:- occur due to mycobacterial hypersensitivity

Three characteristic findings:

i) Tubercular test (Montoux test) positive: due to hypersensitivity
ii) Lesions are AFB negative i.e. acid fast bacilli/mycoplasma infection is absent.
iii) ATT → patient respond well to Antitubercular Therapy.

Types of Tuberculids:

1. **Micronodular / lichen scrofulosorum**
- tiny, lichenoid papules over trunk commonly seen in children.

2. **Papulonecrotic**
- multiple papules with central necrosis usually heal with scarring seen in extremities

3. **Erythema Induratum of Bazin/ Bazin disease**
- Erythematous nodules develop over posterior leg usually ulcerate.
- Lobular panniculitis

Important points:

I) Facultative Tuberculid/ Erythema nodosum
- Erythema nodosum develop over anterior leg do not ulcerate.

2) False tuberculid/LMDF
- Papules develop over 1/3rd of face (eyelids)

Treatment:

Antitubercular Therapy (for 6 months)

2 months → HRZE

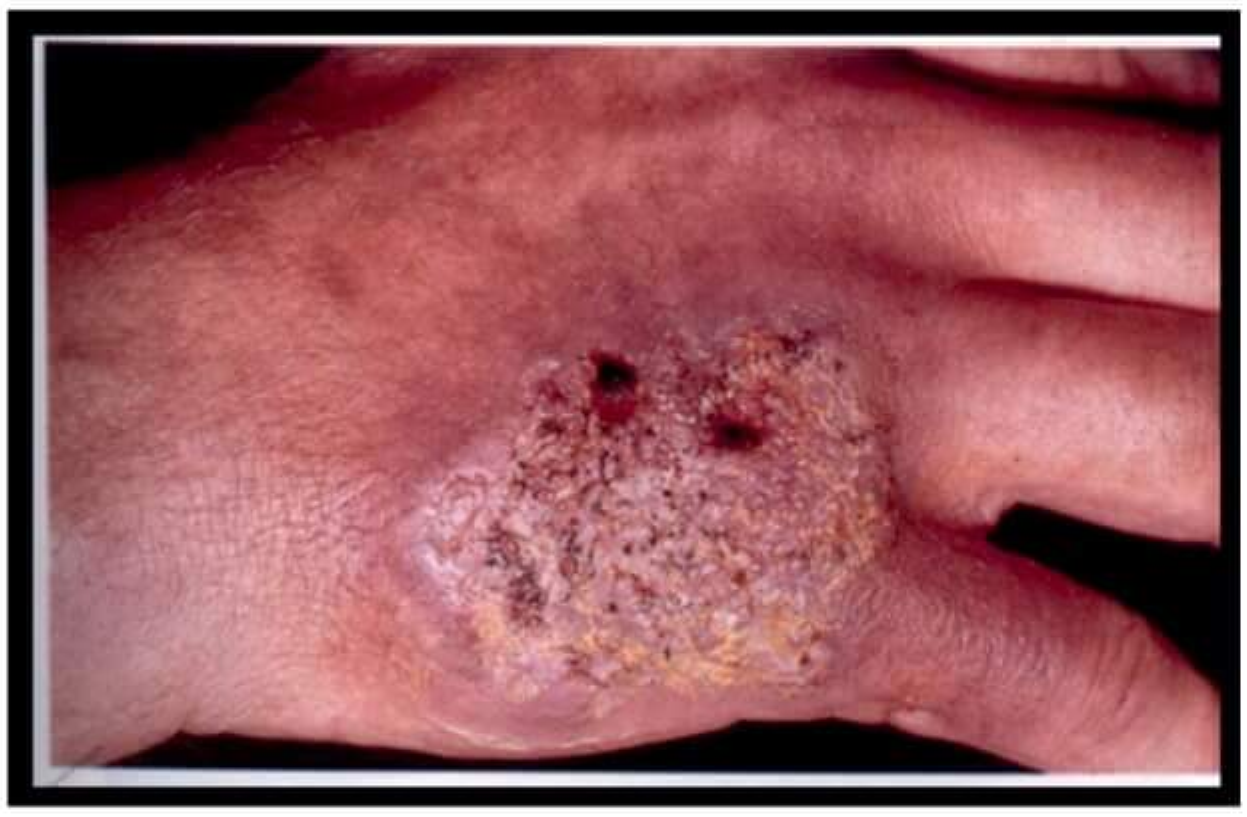
4 months → HRE

Mnemonic

H-Isoniazid (INH)
R-rifampicin
Z- pyrazinamide
E-ethambutol

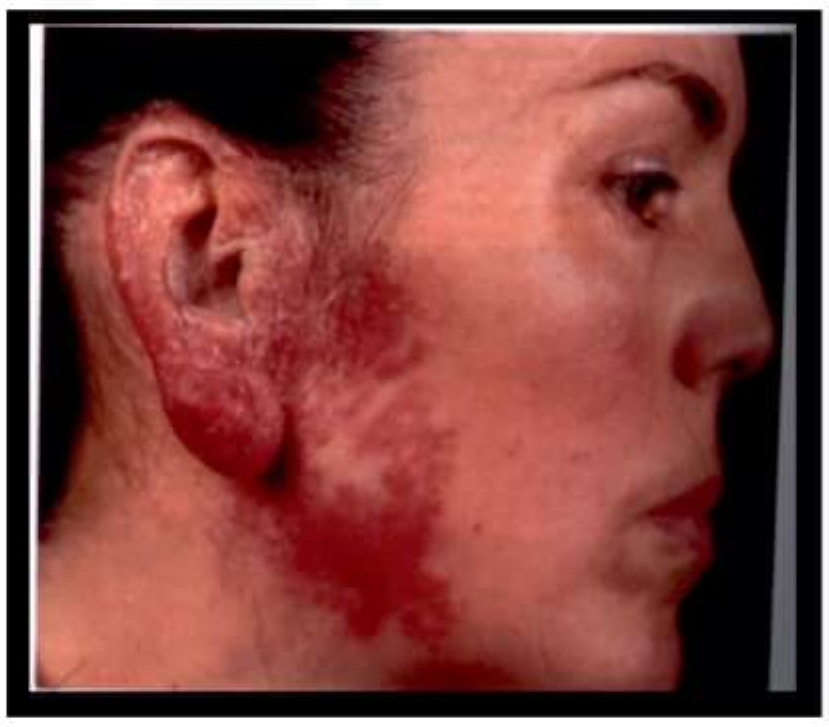
	I	R	E	S	P	(Adult ATT drugs)
For	↓	↓	↓	↓	↓	
Dose	5	10	15	15	25	mg/kg/Body weight

Tuberculosis verrucose cutis (TBVC) or anatomist wart

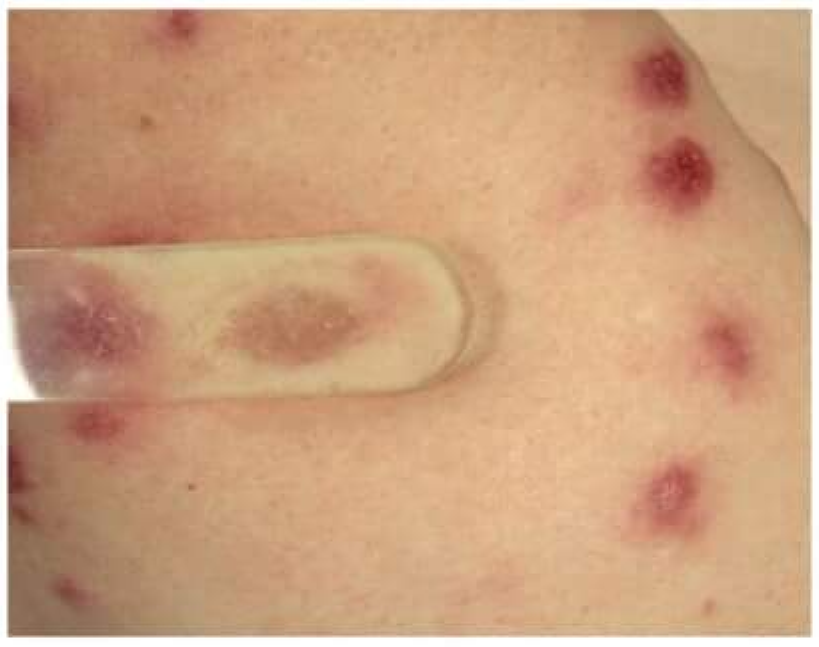


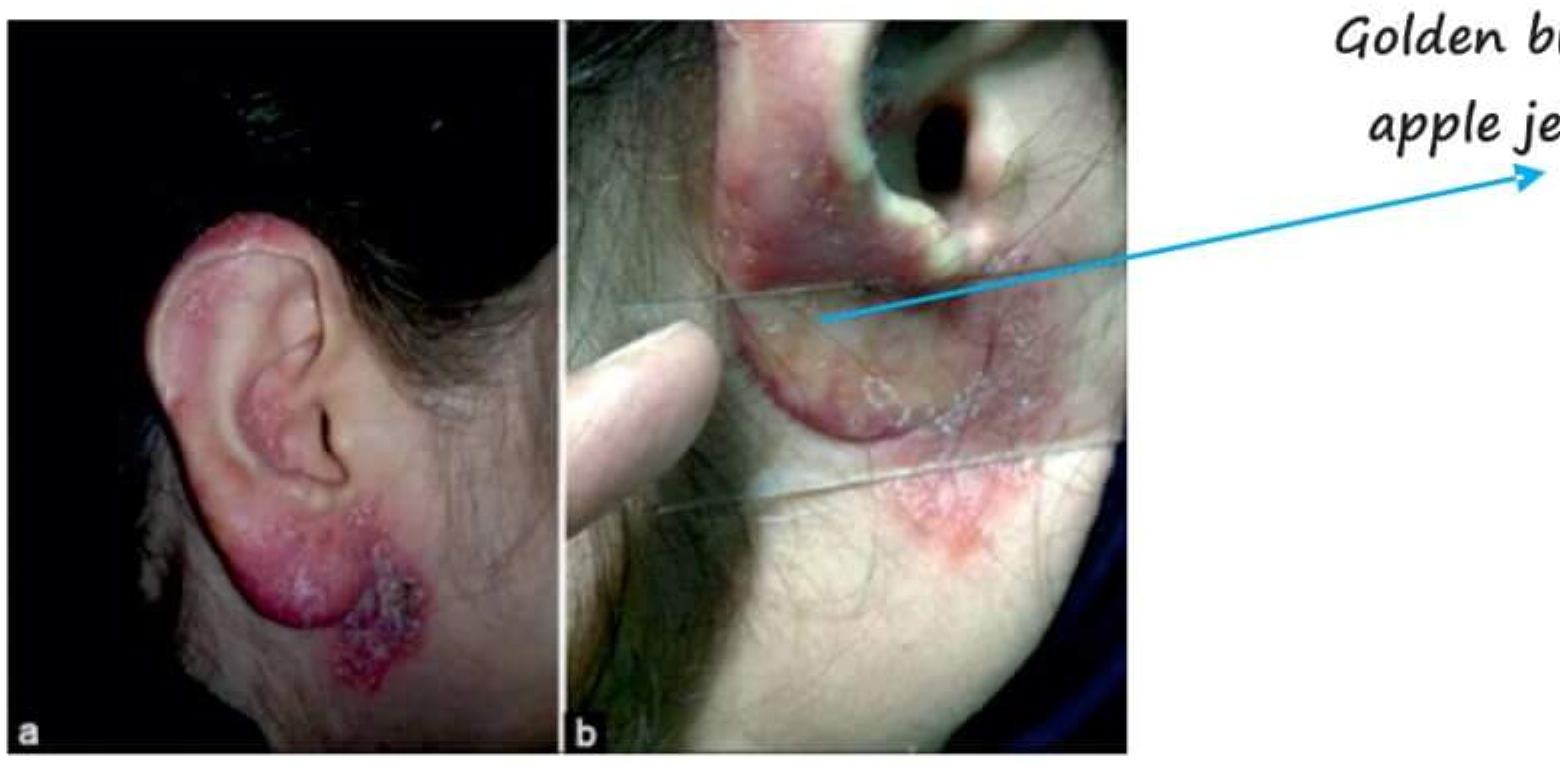
- → Rough verrucous warty lesion usually heals with scar.
- → Generally present on extremities.

Lupus vulgaris



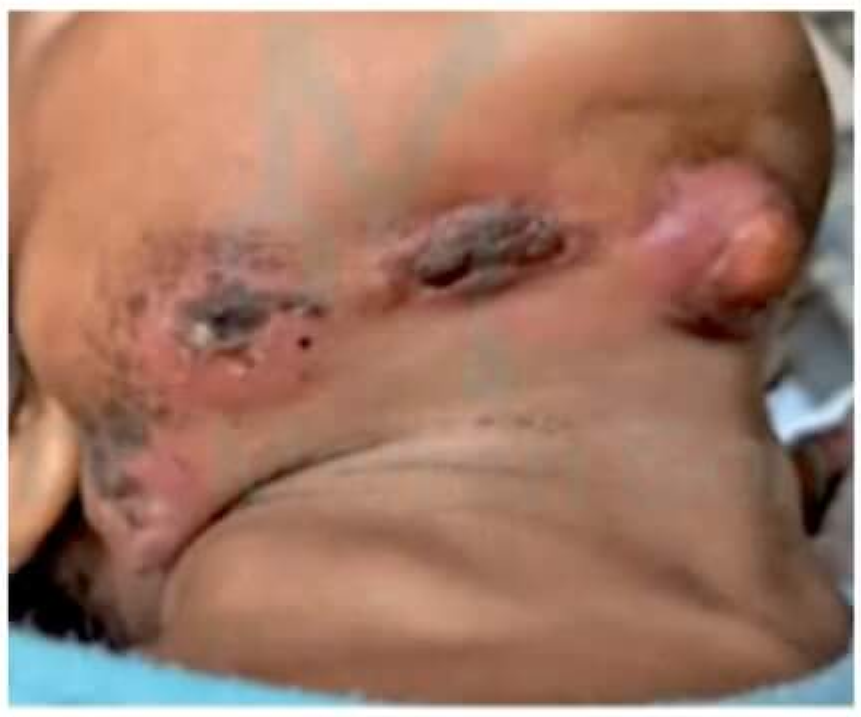
- Asymmetrical lesions- active margin on one side and scarring on the other side.
  Apple jelly nodules on diascopy- Erythematous lesions will turn from pink to golden brown.



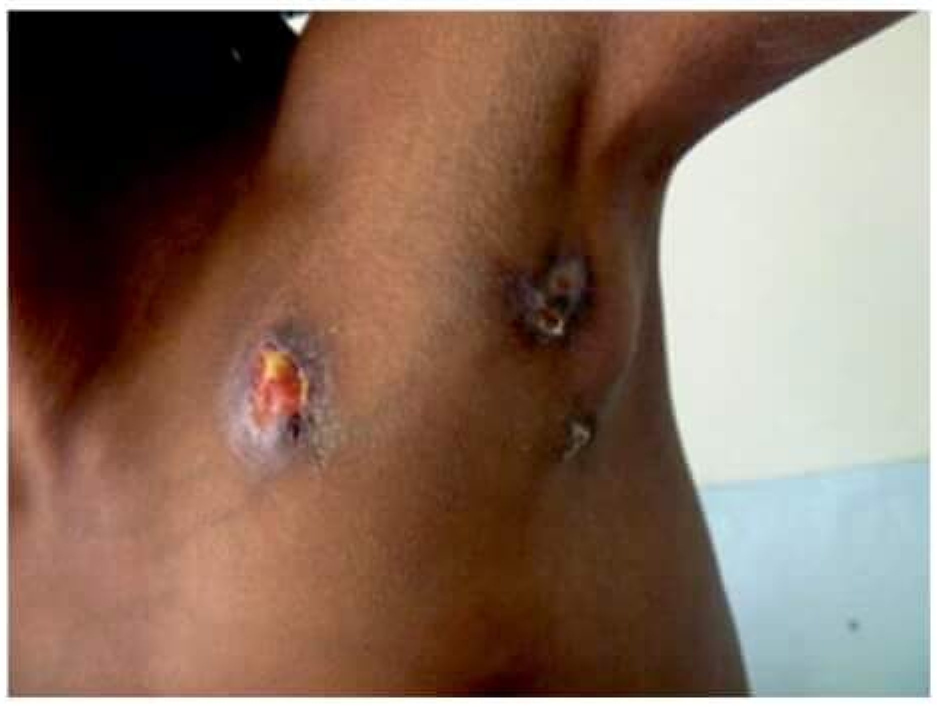


Golden brown color
apple jelly nodules

Scrofuloderma over neck



- → Most common in children over neck area.
- → Cervical lymph nodes are most commonly involved.
- → Enlarged lymph nodes ruptured to form multiple sinuses with discharge.'



- → Axillary involvement present in the above image

Important Fact : -

Atypical mycobacterium

M.marium
↓
Swimming pool granuloma[Q]/
Fish tank granuloma

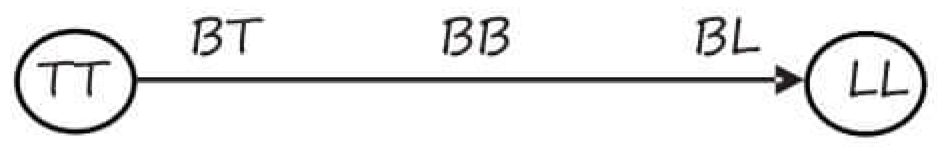
M.ulcerus
↓
Buruli ulcer[Q]

I. **Leprosy**: - caused by mycobacterium leprae

a. **According to Ridley Jopling classification**

There are 5 types depending on the host immunity:

Determinate types:



TT, LL – Stable
BB, BB, BL- Unstable
BB- rarest /most unstable

There are 5 types:

- TT - Tubercular
- BT - Borderline Tubercular
- BB - Borderline Borderline
- BL - Borderline Lepromatous
- LL - Lepromatous

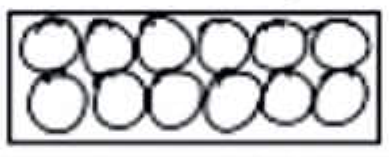
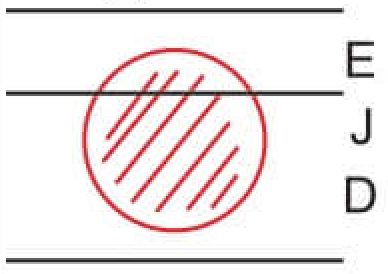
Indeterminate types of leprosy:

→ Self heal or may persist in the same way or can deteriorate.

Pityriasis alba		Indeterminate Leprosy
1)single	+	+
2)Face	+	+
3)Child	+	+
4)H/O Atopy	+	–
5)Scaling	+	–
6)Sensation intact	+	–
7)Atrophy	–	+
8)Histo:Spongiosis		Peri -appendageal, perineural lymphocytic infiltrate.
9)Treatment:Self limiting		PB -MDT

a. According to Ridley Jopling classification

		TT → LL
Bacteriological	1.Bacterial Load	→ ↑
Immunological	2. cell mediated immunity	→ ↓
	3. Lepromin test (marker of cell mediated immunity) Positivity	→ ↓
	4. CD4: CD8::2:1	Reversed
	5. inflammatory mediators	IL 2,12, INFγ → IL-5,10

Clinical	6. lesions 7. symmetry	single → multiple Asymmetric → Symmetric
Histological	8.findings	TT Epitheloid Granuloma with giant cells & occupied Absent grenz Zone. LL Foam cells within globi (lipid laden macrophages) - Free /Present grenz zone - cigar or Cigarette Arrangement of foam cells is known as globi  Grenz zone: clear, uninfiltrated zone in upper dermis  TT-cell mediated Immunity-good ↓ LL

Important Facts:

1. Most common type : Borderline Tuberculid
2. Rarest Type : borderline borderline(BB)
3. MC cranial nerve : facial
4. MC peripheral nerve : ulnar> Post. Tibial
5. 1st sensation lost in leprosy : Temperature (cold>Hot)
6. Satellite lesions : Borderline Tuberculid
7. Punched out/inverted saucer shaped/ swiss cheese appearance Borderline Borderline
8. Blindness in leprosy : Exposure keratitis
9. Virulence of M.leprae : Phenolic Glycolipid I
10. Doubling time of this bacteria- 11-14days
11. Other Types
    1. **Pure neuritic type: -**
        - → Skin involvement absent
        - → Skin smear is negative
        - → Nerve involvement present

          Nerve biopsy- I) sural nerve

          ii) Cutaneous branch of radial nerve
    2. **Histoid leprosy**
        - → Multiple nodules seen over normal appearing skin
        - → Commonly seen after dapsone monotherapy
    3. **Lucio leprosy/Lepra bonita/beautiful leprosy**
        - → Diffuse skin infiltration makes it erythematous, smooth and shiny.
    4. **Lazarine Leprosy**
        - → seen in malnourished individuals.
        - → Ulcerative lesions present.

LEPRA REACTION

	Type I/reversal reaction	Type II/ENL reaction
1. Hypersensitivity	IV	III
2. Leprosy →	Borderline tubercular Borderline Borderline, Borderline lepromatous	Borderline lepromatous lepromatous
3. Inflammatory mediator →	IL-1,2 INF-γ	TNF-α^{Q} IL-4,5
4. Clinical features –	New lesion: absent Edema	New lesion present Nodular erythema nodosum Leprosum (ENL)
5. Drug of choice →	Steroid	steroid
	Prednisolone 1mg/kg/bwt for 2 weeks & then gradually tapered	
	Thalidomide (Not effective)	**Thalidomide** **(gold standard) best acting,** (since has lot of side effects it is not Drug of choice)

Lucio phenomenon:

- → common in Mexico.
- → Ulcerative lesions are seen over lower extremities which do not respond to thalidomide, so in severe cases exchange transfusion is done.

b. According to WHO classification: are of 2 types based on 3 cardinal features :-

2 types

Cardinal features	Paucibacillary	Multibacillary
i. Hypopigmented/ hypoanaesthetic patches	1-5	>5
ii. Peripheral nerve thickening	0	≥ 1
iii. Skin slit smear (AFB)	Absent	Present

Treatment:

	Multi Drug Therapy (MDT)	
Medicines	Paucibacillary	Multibacillary
1. Rifampicin	600mg/month	600mg/month
2. Dapsone	100mg/daily	100mg/daily
3. Clofazimine	300mg/month 50mg/daily	300mg/month 50mg/daily
Duration	6 months	12 months

In Children 10 to 14years

	Multi Drug Therapy (MDT)	
Medicines	Paucibacillary	multibacillary
1. Rifampicin	450mg/month	450mg/month
2. Dapsone	50mg/daily	50mg/daily
3. Clofazimine	150mg/month 50mg/daily	150mg/month 50mg/daily
Duration	6 months	12 months

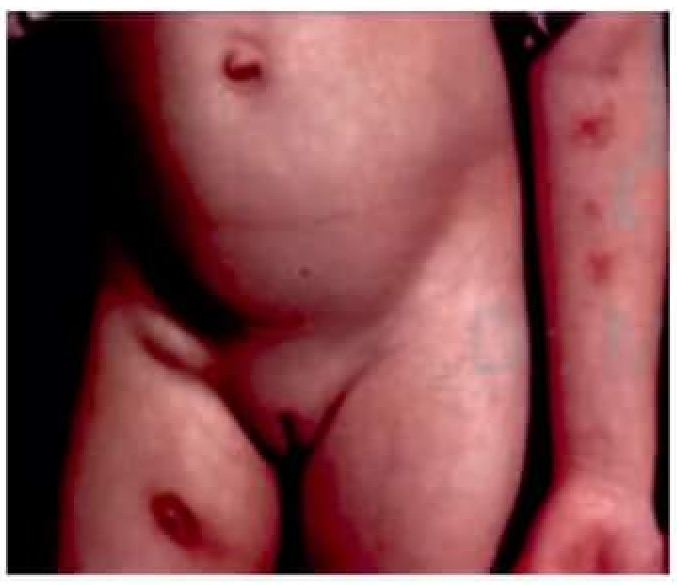
In Children <10 or weight <40kg

→ Rifampicin 10mg/kg/B wt/month
→ Dapsone 2mg/kg/Bwt/ daily
→ Clofazimine → 6mg/kg/Bwt/month
→12mg/kg/Bwt/ daily

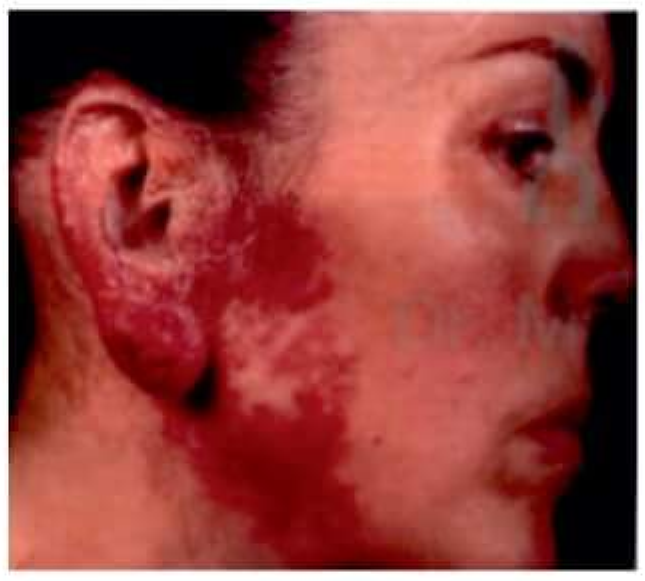
WHO Definition of RFT(Relief from treatment)

PB- 6months in 9months duration
MB-12months in 18months treatment.

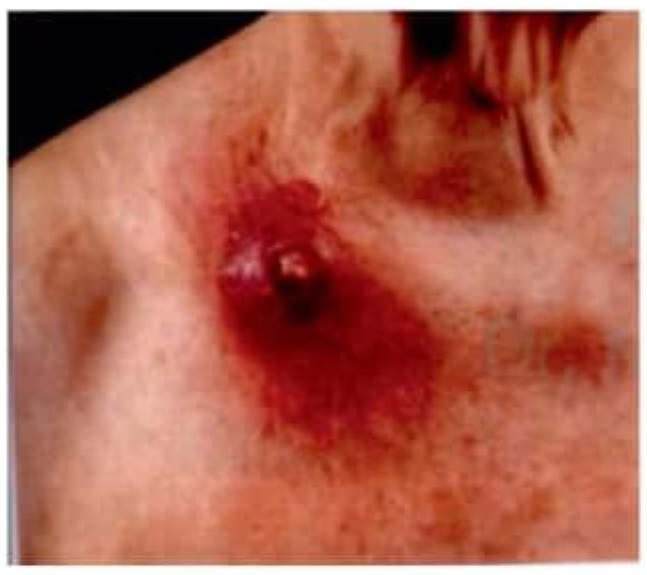
Mycobacterial infections



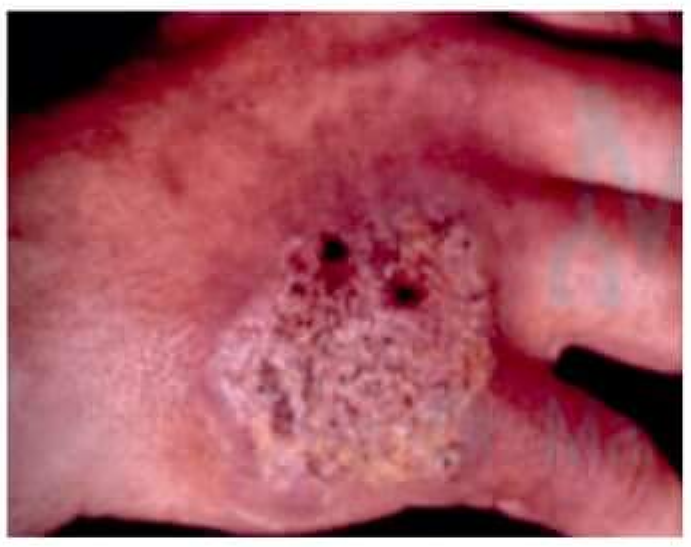
Exogenous-tuberculous chancre



endogenous- lupus vulgaris



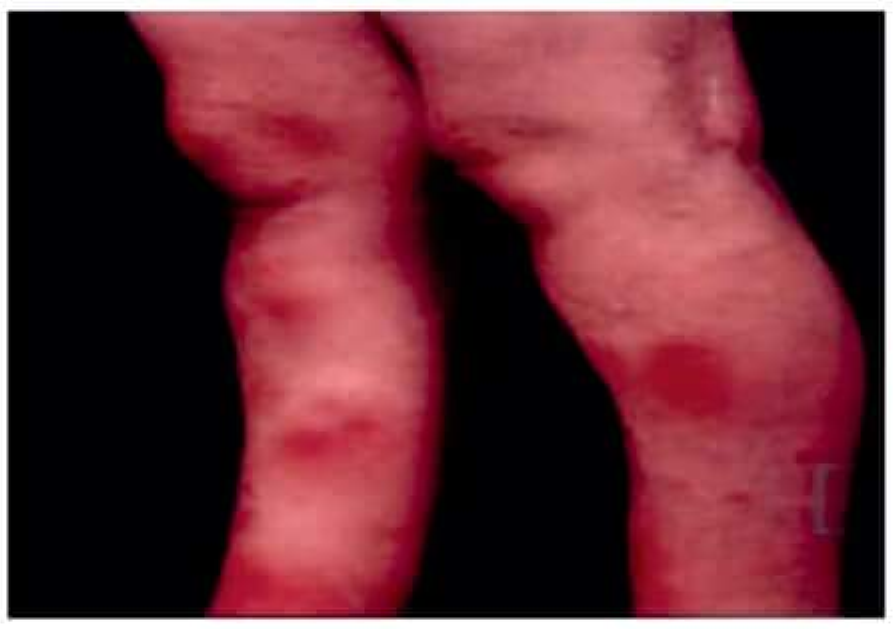
scrofuloderma



Tuberculosis verrucous cutis

	warts	anatomist wart
number	multiple	single
Induration	absent	present
scarring	absent	present (in centre)
discharge	absent	present (serous)

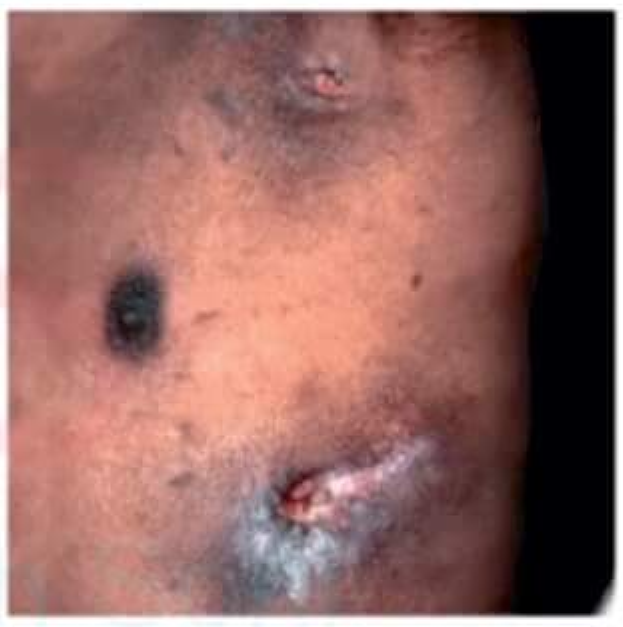
mnemonic: ISD number



Erythema nodosum

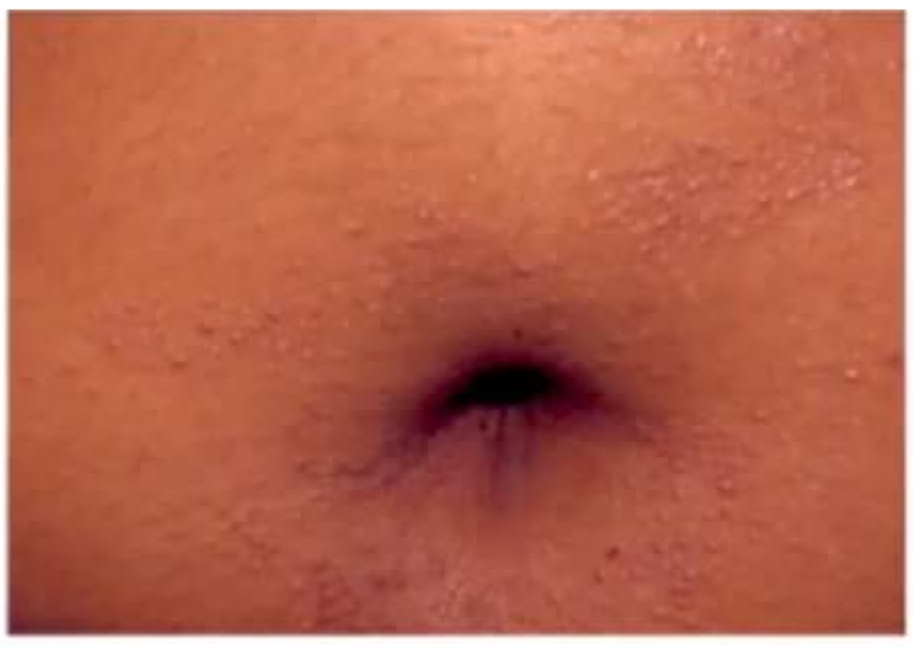
-Bilateral, also seen in Sarcoidosis, Bechets, drugs, streptococcal throat infection.

22). Two ulcers on the chest wall and axilla, associated with underlying sinus tracts and discharge. Diagnosis?

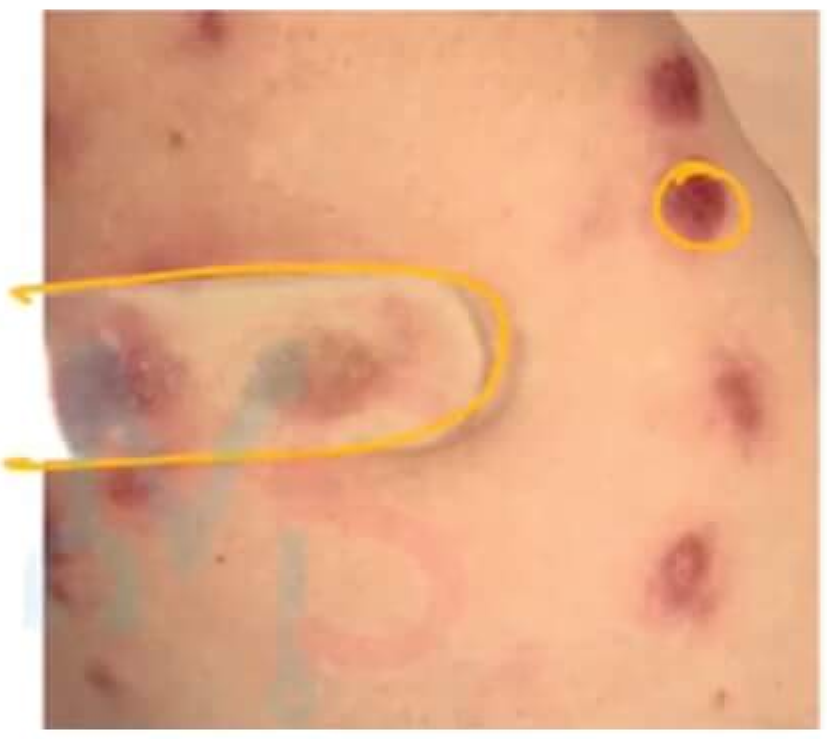


A. Lupus vulgaris
B. Scrofuloderma
C. Lichen Scrofulosorum
D. TBVC

22. ANS. B

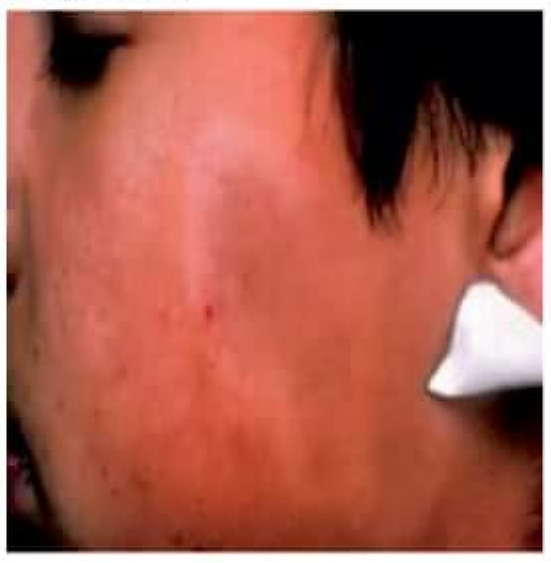


Tiny lichenoid papules over trunk-
Lichen Scrofuloderma

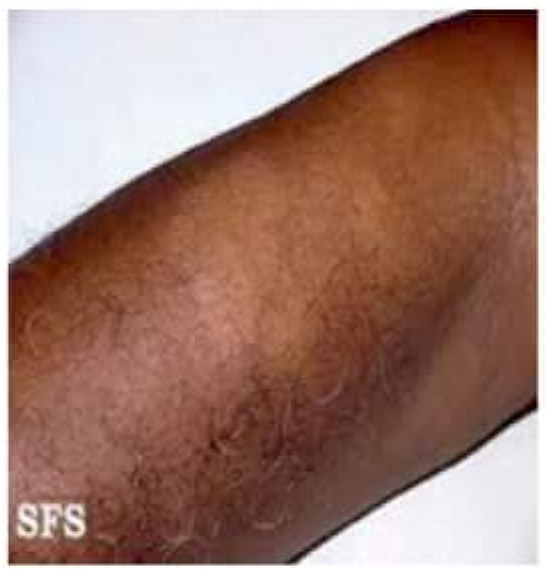


Apple jelly nodules

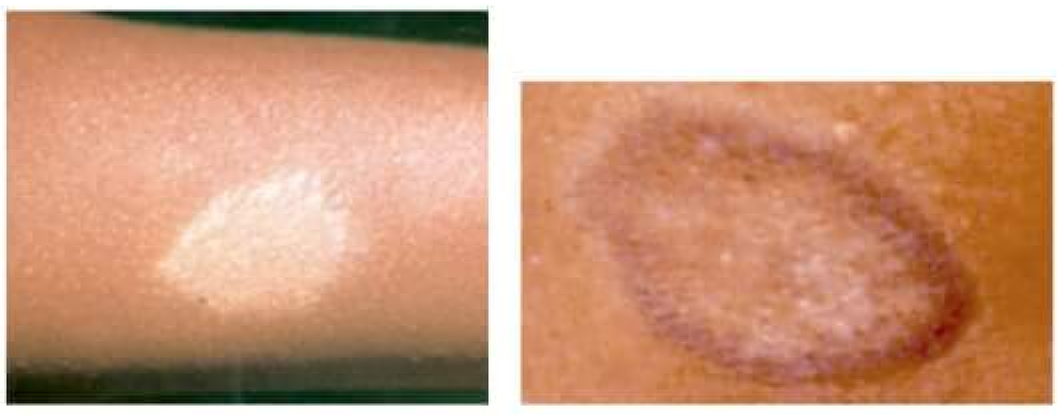
Leprosy



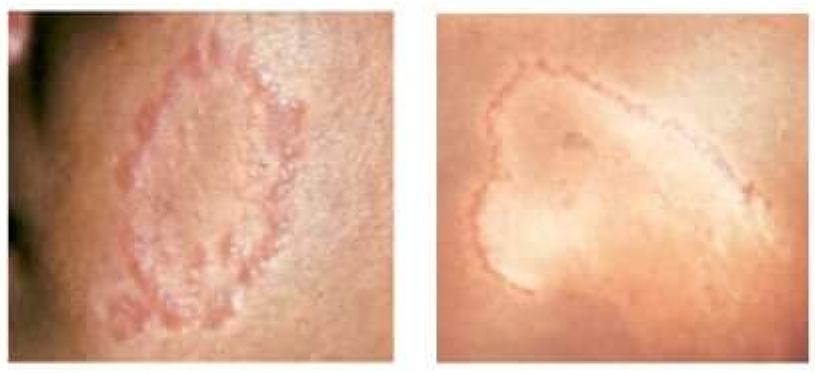
Indeterminate leprosy



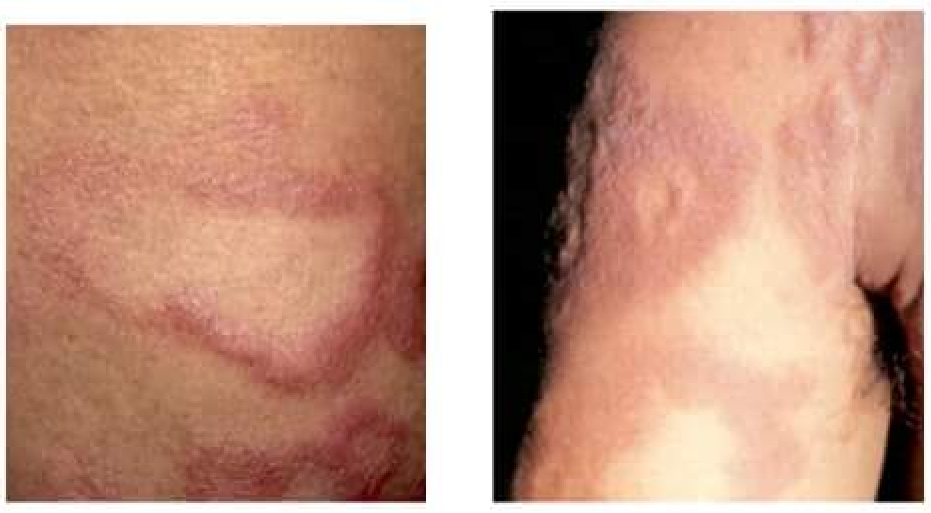
Tuberculoid



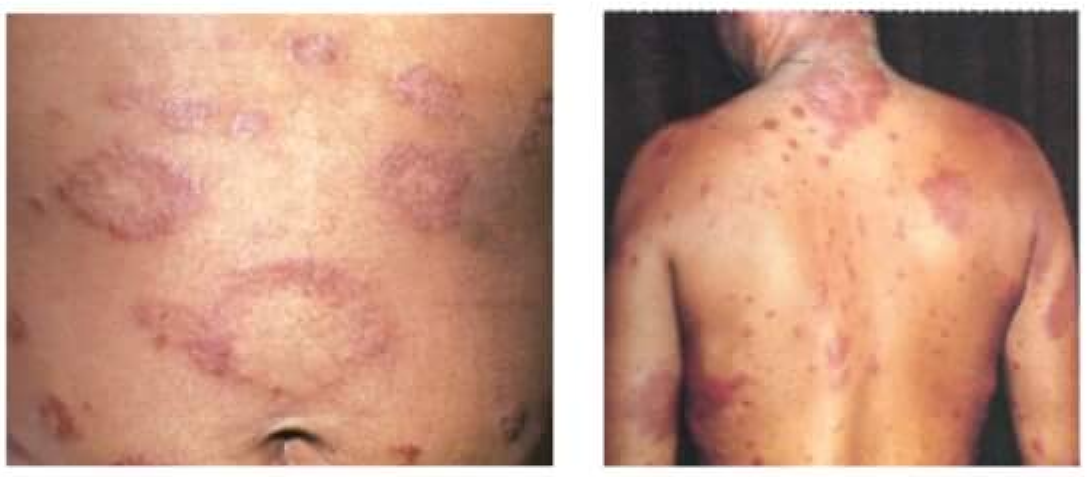
utberculoid



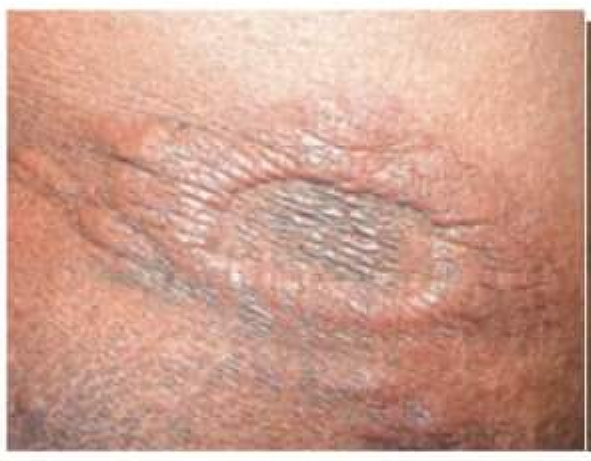
satellite lesions in borderline tuberculid



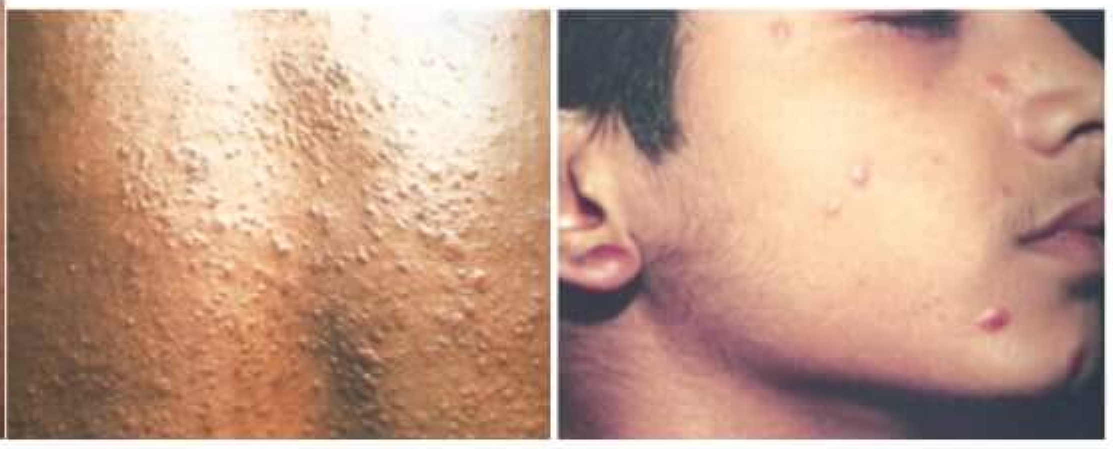
Borderline borderline



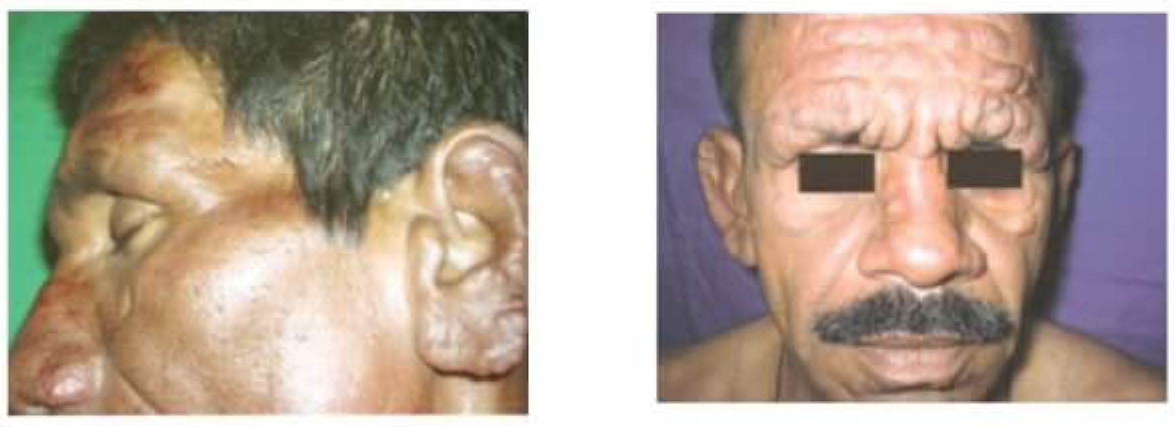
borderline lepromatous



Punched out lesion
Borderline borderline

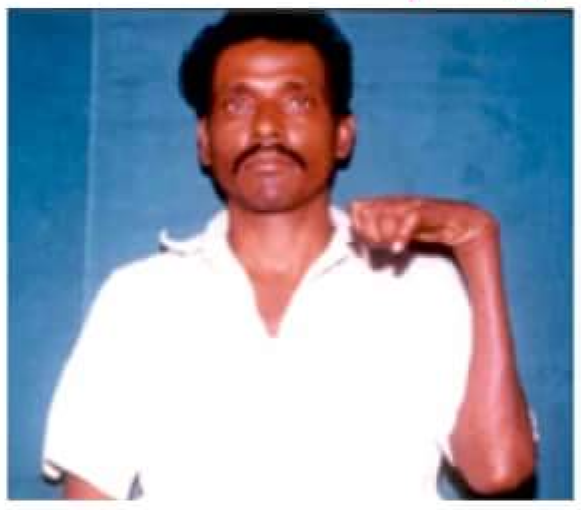


Lepromatous leprosy

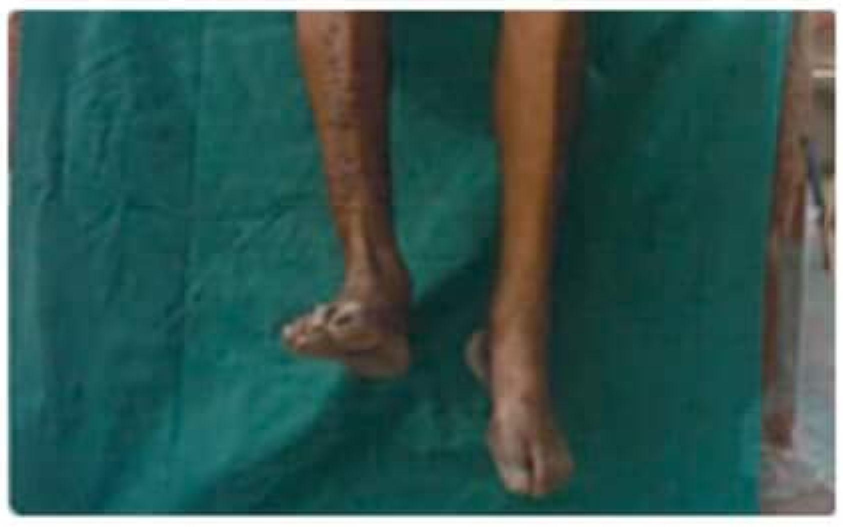


leonine facies (Lepromatous leprosy)

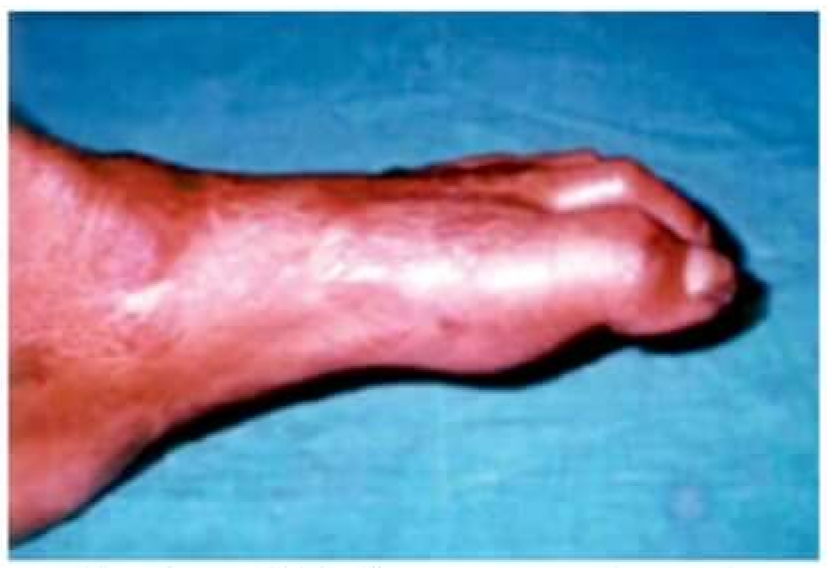
Nerves involved in leprosy



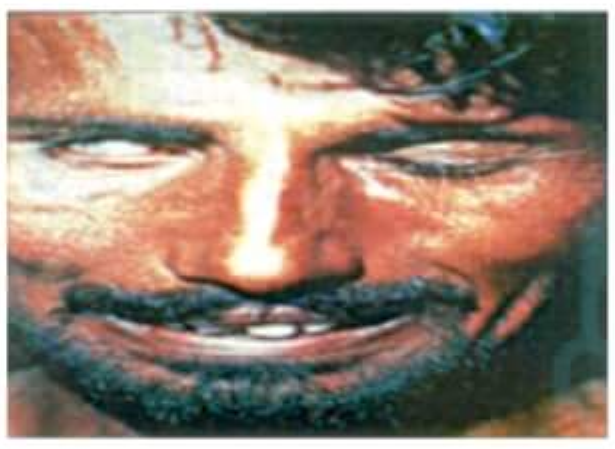
Radial nerve- wrist drop



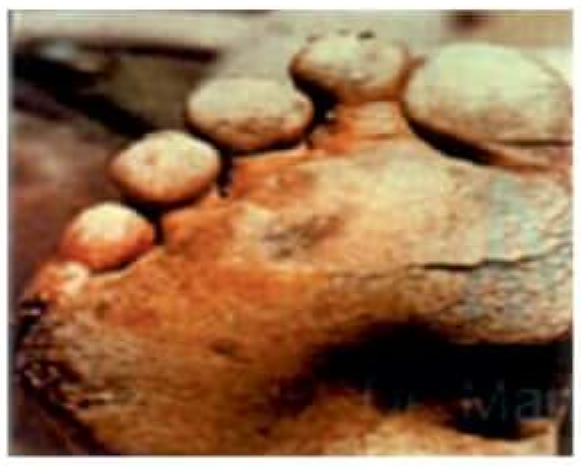
lateral popliteal nerve- foot drop



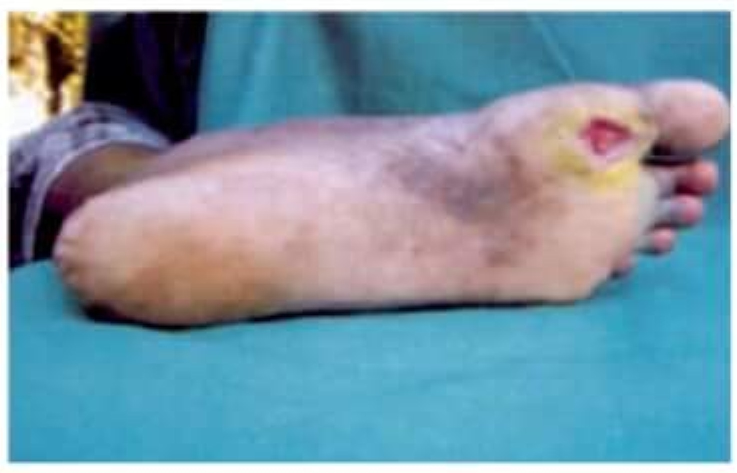
posterior tibial nerve- claw toes



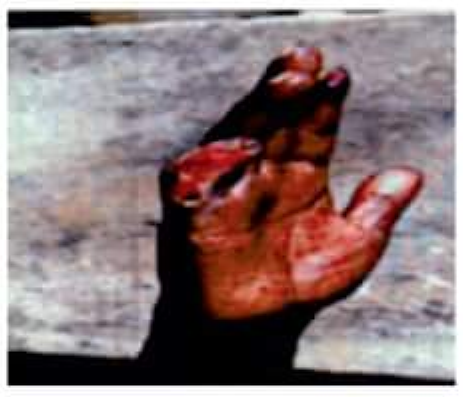
Facial nerve- lagophthalmos
Facial palsy



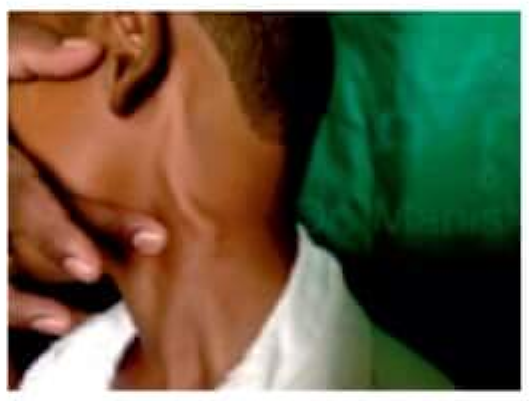
Ichthyosis(fish like scales)



plantar ulcer

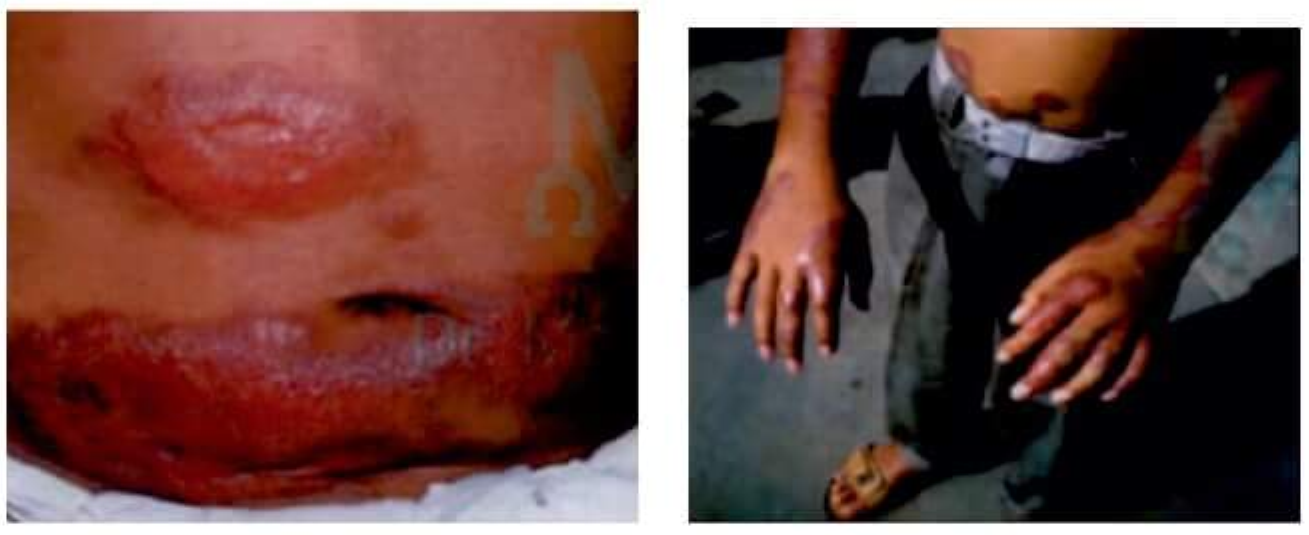


palmar ulcer

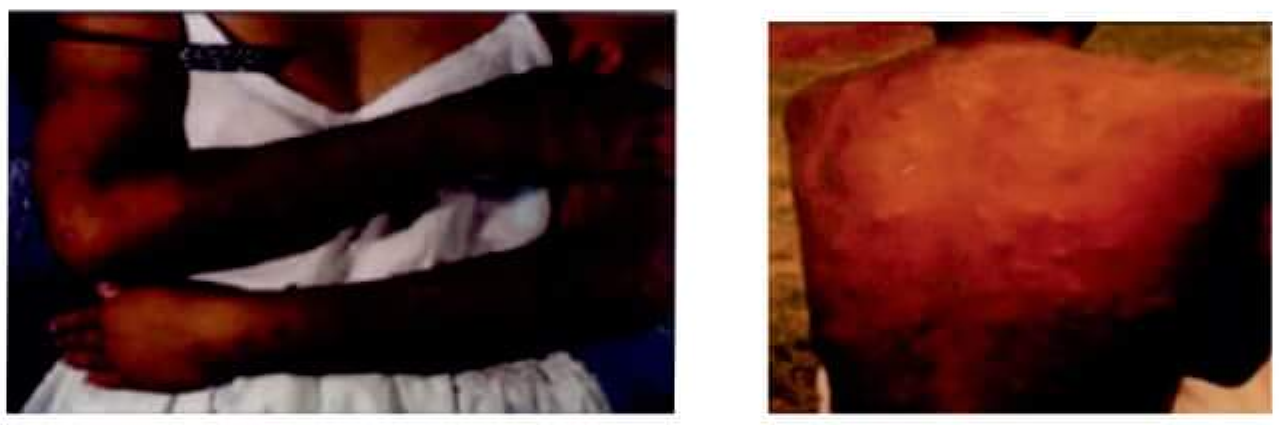


thickened greater auricular nerve

Lepra reaction I



Lepra reaction II/ENL reaction -type 3 HSN



Nine banded armadillo (for culture of M.leprae)

VIRAL INFECTION

1) WARTS

- Caused by HPV

Types	HPV Subtypes
1. Deep plantar wart (myrmecia)	1 (MC)
2. Superficial plantar wart (mosaic)	2 (MC)
3. Plane wart (Verruca plana)	3, 10
4. Common wart (V. Vulgaris)	4 < 2 (MC)
5. Epidermo dysplasia verruciformis	5, 8
6. Laryngeal wart / Anogenital wart	6 > 11
7. Butcher w Mnemonic "DSP -CELB"	7 (MC)

- **Investigations:**

  Histological examination

  KOILOCYTES – keratinocyte with hyperchromatic nucleus

- **Treatment:**

  → Cryotherapy (-196°C liquid nitrogen) – Freezing of Lesions

  → Electrocautery

  → TCA (Trichloroacetic acid) application (70-80%) (CHEMICAL CAUTERY)

Important Facts About STI (Anogenital warts) / Condyloma acuminata

→ Causative organism HPV→ 6 > 11

- **C/F**:

  → Pointed lesions

  → Asymptomatic

  → Pink verrucous lesions

- **Site:**

  In Males – Coronal sulcus, Frenulum

  In Females – Posterior Fourchette

- **Treatment** - Topical {Imiquimod > Podophyllotoxin (PODOPHYLLIN)}
- **Mechanism of action:**

  1) Imiquimod – Acts on toll like receptors 7&8 and improves the immunity, therefore it's an agonist

  - Both agents are topical agents and teratogenic

#Treatment of Anogenital wart in pregnancy: Cryotherapy > TCA application

#Treatment of Giant Anogenital Warts (Buschke – lowensteintumor): Surgery

2. MOLLUSUM CONTAGIOSUM

→ MCV (Type I-IV) **MC → Type I**

→ **MC in HIV positive → Type II**

→ MCV is a type of POX VIRUS

Clinical features:

→ Asymptomatic

→ Pink Papules with central umbilication

Site: Children – Face

Adult – Genital (STI)

Investigation:

Histology → H.P Bodies (Henderson Paterson)

(Intracytoplasmic eosinophilic inclusion bodies)

Treatment:

→ Cryotherapy

→ Electrocautery

→ TCA application

→ Needle extraction

3. HERPES

1. Herpes labialis

– Caused by HSV – 1

Clinical features:

Group vesical around lips

Which heal so Virus dormant in nerves / ganglion

Reactivation of virus Causes → Fever blisters/ cold sores seen

Investigation:

→ Tzanck smear: Multinucleated giant cells

Treatment

→ Acyclovir 400 mg 3 times / days X 7 days or 200 mg 5 times / day X 7 days

2. Chicken Pox

→ Caused by varicella zoster virus

Clinical feature:

→ Constitutional symptoms → Fever (Mainly in children)

→ Malaise

→ Centripetal Rash

→ Dew drop on Rose Petal

- **Treatment:**
  - → Acyclovir 800 mg QID X 7 days

3. Herpes Zoster

→ Reactivation of varicella zoster virus

- **C/F** – Unilateral grouped vesicles along particular dermatome
- **Treatment – DOC is Acyclovir 800 mg 5 times / day X 7 days**
- **Complication**
  - → Post – herpetic neuralgia

↓

Neuralgic pain for > 4 weeks persists (preferably 3 months) at the site where lesions of herpes zoster are healing

Hutchinson's sign

→ Herpes ophthalmicus

→ If lesions on the Tip of nose suggestive of "eye involvement"

- **Treatment**
  - → Pregabalin
  - → Gabapentin

Treatment of post herpetic neuralgia

4. STI (Herpes Genitalis)

→ Caused by HSV II (around genitals)

- **C/F** – Grouped vesicles rupture to form → Polycyclic erosions
- **Investigations** – Tzanck smear – Multinucleated giant cell

- **Treatment**
  - → Acyclovir
    - 400 mg, 3 times a day
    - Or
    - 200 mg, 5 times a day

X 7 days

Note: To differentiate CORN from WARTS on the plantar surface we have to remove the top most layer, in warts on removing there is blood oozing out whereas in Corn there is absence of blood

Extra edge:

ECZEMA HERPERTICUM – It's a disseminated viral infection characterized by fever and clusters of itchy blisters or punched out erosions. It is most often seen as a complication of ATOPIC DERMATITIS/ECZEMA. Mainly seen in patients with eczema and develops 2° HSV infection

SEXUALLY TRANSMITTED INFECTIONS

	Syphilis	LGV	Chancroid	Donovanosis
Causative organism	Treponema Pallidum	Chlamydia Trachomatis L1, 2, 3 (MC)	Haemophilus Ducreyi	Klebsiella Granulomatis (Calymmatobacterium granulomatis)
Incubation	9-90 days	3-30 days	2-5 days	8-80 days (till 1 year)
Genital ulcers	1° syphilis			
A) Number	single	Single	Multiple	Single
B) Base	Non-vascular	Non-vascular	Granulomatous & Bleeds	Beefy red, Bleeds Very easily
C) Induration	Very firm	Transient firm	Soft (Absent)	Firm
D) Margins /edges	Elevated	Elevated	Undermined ragged	Elevated
E) Pain	Painless	Painless	Painful	Painless
Lymphadenopathy	→ Bilateral → Nontender → Shotty (rubbery)	→ U/L (2/3rd cases) → tender → Bubo's (without concomittent genital ulcers)	→ U/L → Tender → Bubo's (With Genital ulcers)	Pseudo Bubos (subcutaneous nodule) ↓ Inguinal area (No lymphadenopathy)
Investigations	Dark ground microscopy (D/G, M/S)	Nucleic Acid Amplification Test (NAAT)	1. Gram Staining – G negative Coccobacilli ↓ School of fish Or Rail Road 2. Culture Mueller Hinton media Or 5% chocolatized horse blood agar	Smear ↓ Donovan bodies ↓ Closed safety pin appearance
DOC	Benzathine Penicillin Except for Neurosyphillis: Aqueous / crystalline penicillin	Doxycycline	Azithromycin	Azithromycin
2nd DOC	Doxycycline (Non pregnant)	Azithromycin	Ceftriaxone	Doxycycline

Important Facts:

*FRIE TEST → for LGV
ITO TEST → Chancroid
} Intradermal test used in past

* LGV → Genital ulcers are transient (Short duration) and painless

Late complications:

1. **Groove sign of green blatt**

   Lymph nodes enlarge on either side of inguinal ligament forming a groove

2. **Genital elephantiasis**

Female	**Male**
↓ leads to	↓ leads to
Estheomine	Saxophone
(eating away of genitals)	Penis (Size increased)

3. **Ano rectal syndrome**
4. **Vagino rectal syndrome**

Types of Donovanosis:

1. **MC → Ulcero granulomatous type (Beefy red)**
2. Hypertrophic type → walnut growth develops
3. Necrotic
4. Sclerotic

Syphilis

Types:

- **Primary syphilis** → Painless, clean based single with firm indurated ulcer & rubbery bilateral non tender lymphadenopathy
  DOC -> Benzathine Penicillin
- **Secondary syphilis** → Rashes, all kinds of lesions can be seen here except vesiculobullous lesions
  → Pigmented lesions on palm and sole
  → Shows deep dermal tenderness: BUSCHKE OLENDORFF SIGN
  – Condyloma lata **(condyloma accuminata by warts in viral infection)**→ Moth eaten alopecia are seen
  → Highly infectious → condyloma lata

  Investigation of Choice – VDRL / RPR
  Drug of Choice – Benzathine penicillin

- **Latent syphilis** → < if 2 years (early)
  → if > 2 years (late)
  } **C/F are absent, serological test are positive**

Investigation of Choice – EIA (Enzyme Immunoassay)

DOC – Benzathine Penicillin

EARLY (in 1°, 2°, early latent)	LATE (in late latent or tertiary syphilis)
↓	↓
BENZATHINE PENICILLIN	BENZATHINE PENICILLIN
↓	↓
2.4mu(1.2MU each buttock IM deep)	2.4 MU(1.2 MU each buttock deep IM injection Weekly for 3 weeks, thus total 7.2 MU)

- **Tertiary syphilis:**

System involvement present as

Gumma
→ Rubbery nodules with Punched out Ulcers Base – washed (Leathery)
→ Tabes Dorsalis

CVS
→ Aneurysm (Ascending aorta)
→ Valvular heart disease (aortic regurgitation)

CNS (Neuro)
→ Generalized paresis of the insane (GPI)
→ Psychiatric problem (Gait abnormality)

Investigation of choice: CSF VDRL
(Look for CSF Pleocytosis as it tells us about treatment)

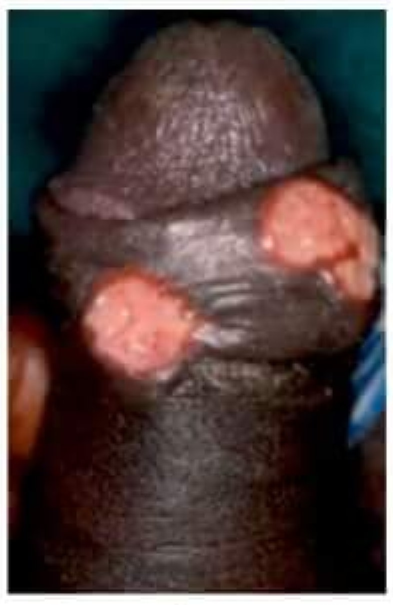
Congenital Syphilis → < 2 years (Early)
→ Vesicobullous lesions
→ Nasal snuffles (Rhinitis)

→ > 2 years (Late):

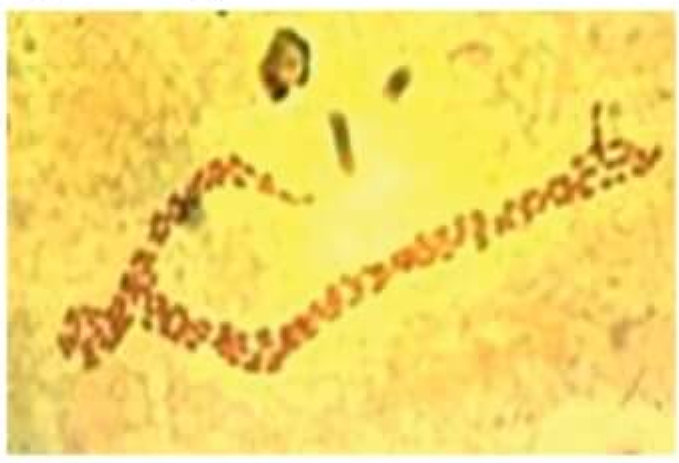
Hutchinson's triad
→ Deafness (VIII nerve Deafness)
→ Interstitial Keratitis
→ Deformed teeth Hutchinson teeth With notch, peg / barrel Shaped

CHANCROID

– The ulcer, ragged, undermined edges, surrounded by a red, very vascular areota

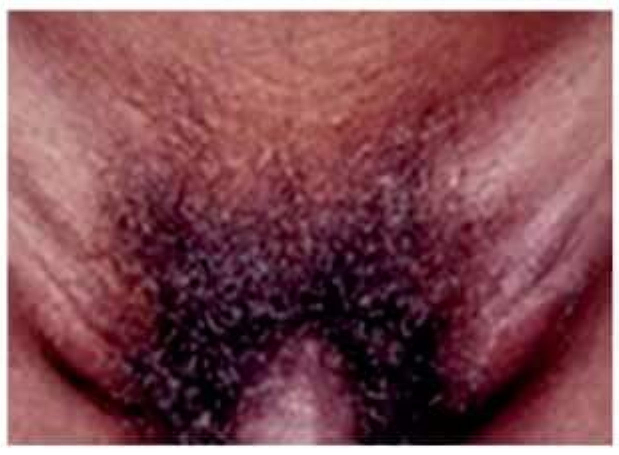


Gram or giemsa staining bacilli in parallel chains of two or three organisms
Streaming along strands of mucus, known as school fish or nail road track



Donovanosis(Granuloma inguinale/ Granuloma venerum)

1. **Ulcer granulomatous**
2. **Hypertrophic or verrucous type**
   (An ulcer or growth with walnut like appearance)
3. **Necrotic**
4. **Sclerotic or cicatricial**

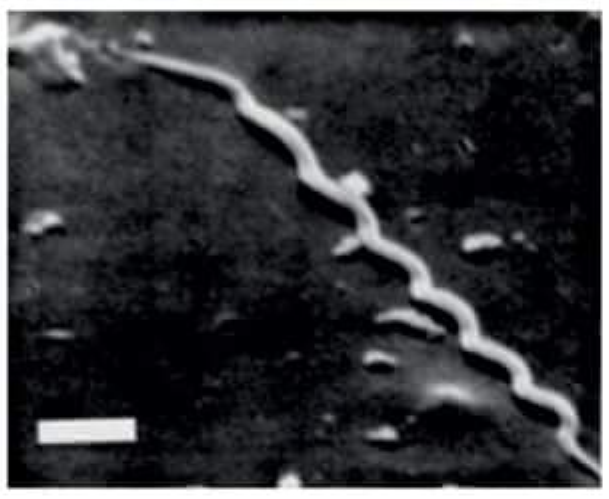


Pseudobubos

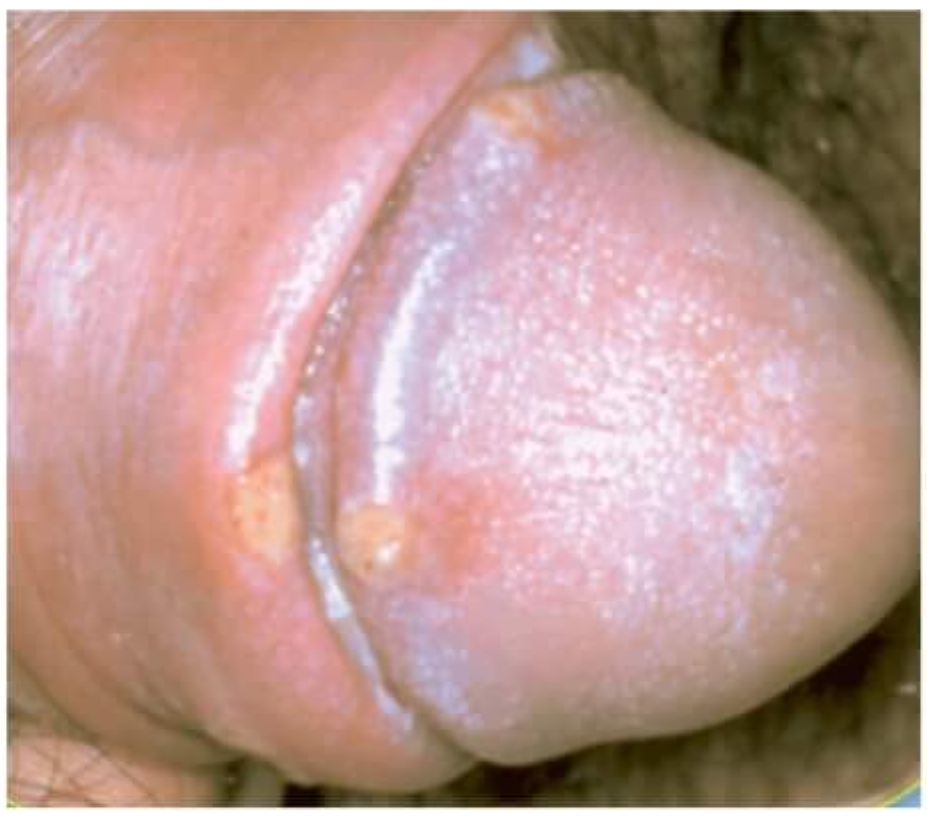
Extra edge:

Anogenital warts: Condyloma accumiata, which have a cauliflower like appearance

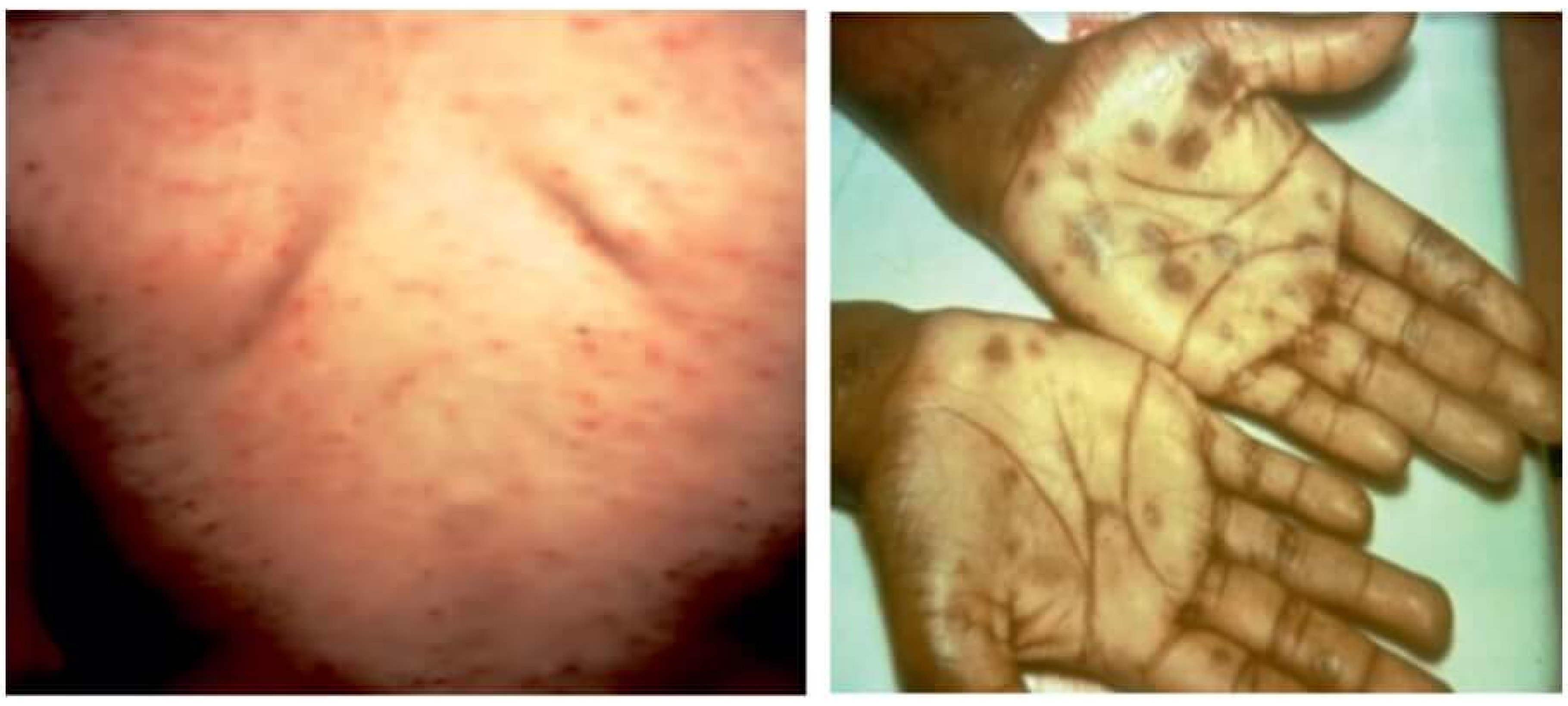
Molluscum Contagiosum: MCV(Molluscum contagiosum virus) is large, brick shaped pox virus that replicates within the cytoplasm of cell



Treponema Pallidum:
Showing spiral and angling on dark field microscopy



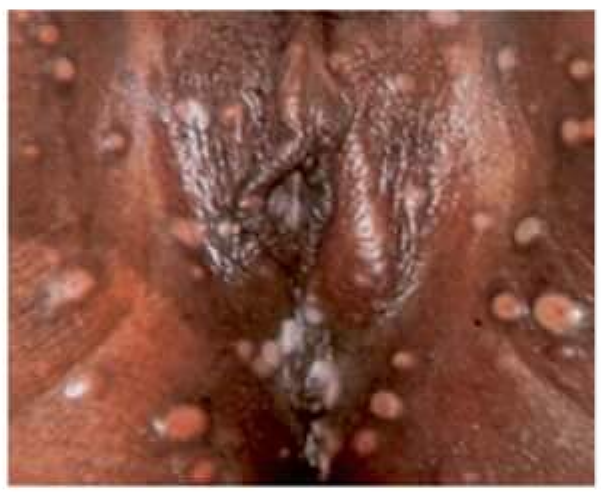
Syphilis chancre (Kissing ulcer)



Secondary syphilis

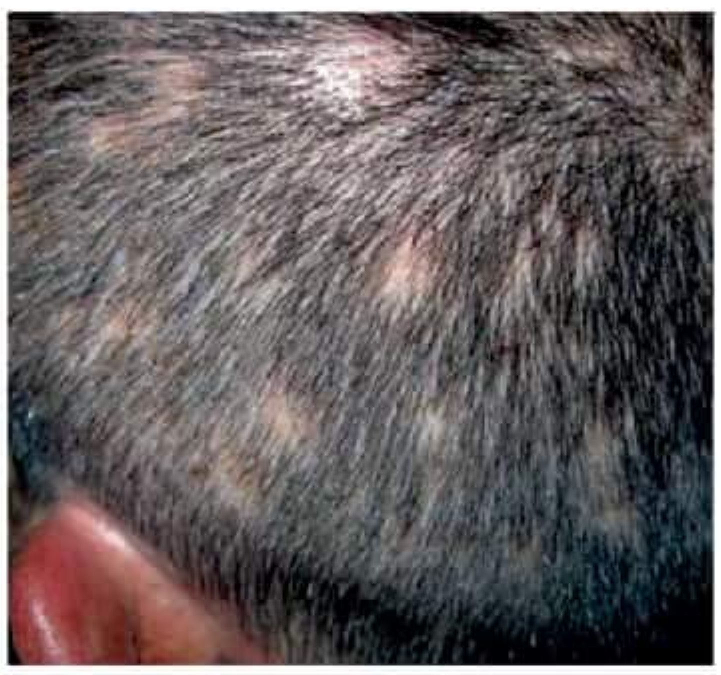
Typical papulosquamous syphilisAnnular lesions seen in syphilis

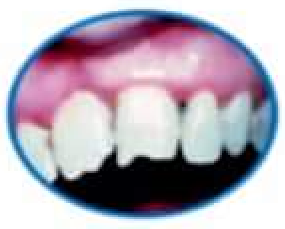
In warm & moist areas, papules may become confluent, hypertrophic & flatted condyloma lata (highly infectious lesions)



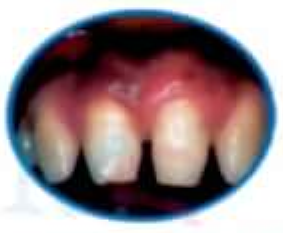
Condyloma Lata

- Patchy, hair loss leaving scattered, irregularly thinned, "moth eaten" patches of semi baldness: **Moth eaten Alopecia seen in secondary syphilis**





Hutchinson's teeth are commonly notched at the free margin



The upper central incision may be peg or barrel-shaped

- JARISCH- HERXHEIMER REACTION: Its an acute febrile reaction, mediated by cytokines that occurs in many patients within 24hr of commencing treatment in patients with syphilis

UrethralDischarge

Causes:

Gonococcal	Non-Gonococcal
Causative agent: - Neisseria Gonococcal	1. Chlamydia Trachomatis 2. Ureaplasma urealyticum 3. Trichomonas 4. Mycoplasma
Incubation Period : 2 - 5days	7-14 days
Clinical feature – Profuse, purulent discharge	Scanty discharge
Investigation → G stain : G negative Diplococci in PMNL (Neutrophils) ↓ Culture – THAYER MARTIN	NAAT (Nucleic Acid Amplification Test)
Treatment → Dual therapy 1. Azithromycin 1 gm stat + 2 Injection ceftriaxone 250 IM stat Or Tab Cefixime 400 mg stat	Treatment → Azithromycin 1 gm stat Or Doxycycline 100 mg BD X 7 days

PAPULOSQUAMOUS DISORDERS

1. Psoriasis

→ Is a chronic inflammatory condition with multiple immunological genetic and environment factors causing erythematous plaque with silvery white (Mica like) scaling.

Predisposing factors

→ HLA CW6 → juvenile psoriasis
→ HLA B27 → psoriatic arthritis
Pustular psoriasis

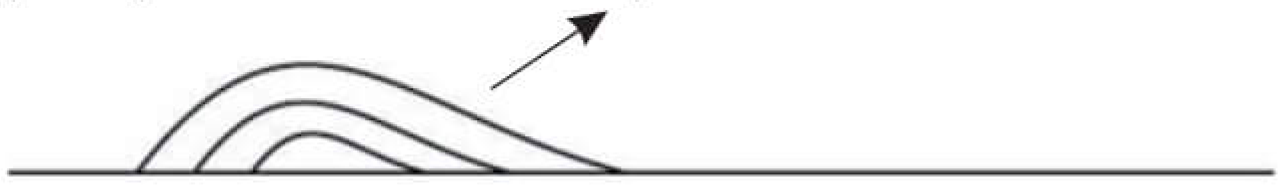
Clinically – various types

i) Psoriasis vulgaris / chronic stationary Type: erythematous plaque with silvery white scaling seen mainly over extensors[Q](knee, elbow, back), scalp, palm & sole

Other isolated involvement.

ii) Scalp psoriasis → only scalp involvement
iii) Palmo plantar → only palm and sole involvement
iv) Inverse psoriasis→ Only flexors[Q]involvement
v) Guttate psoriasis→ In child outer URTI
↓
Means dew drops/ small size lesion[Q]

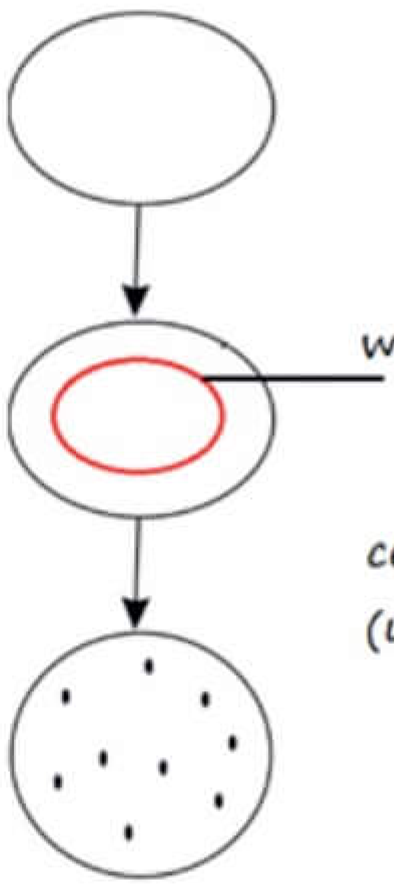
vi) Rupoid psoriasis – limpet like conical scales.



Conical scales adherent to the skin surface

C/f:

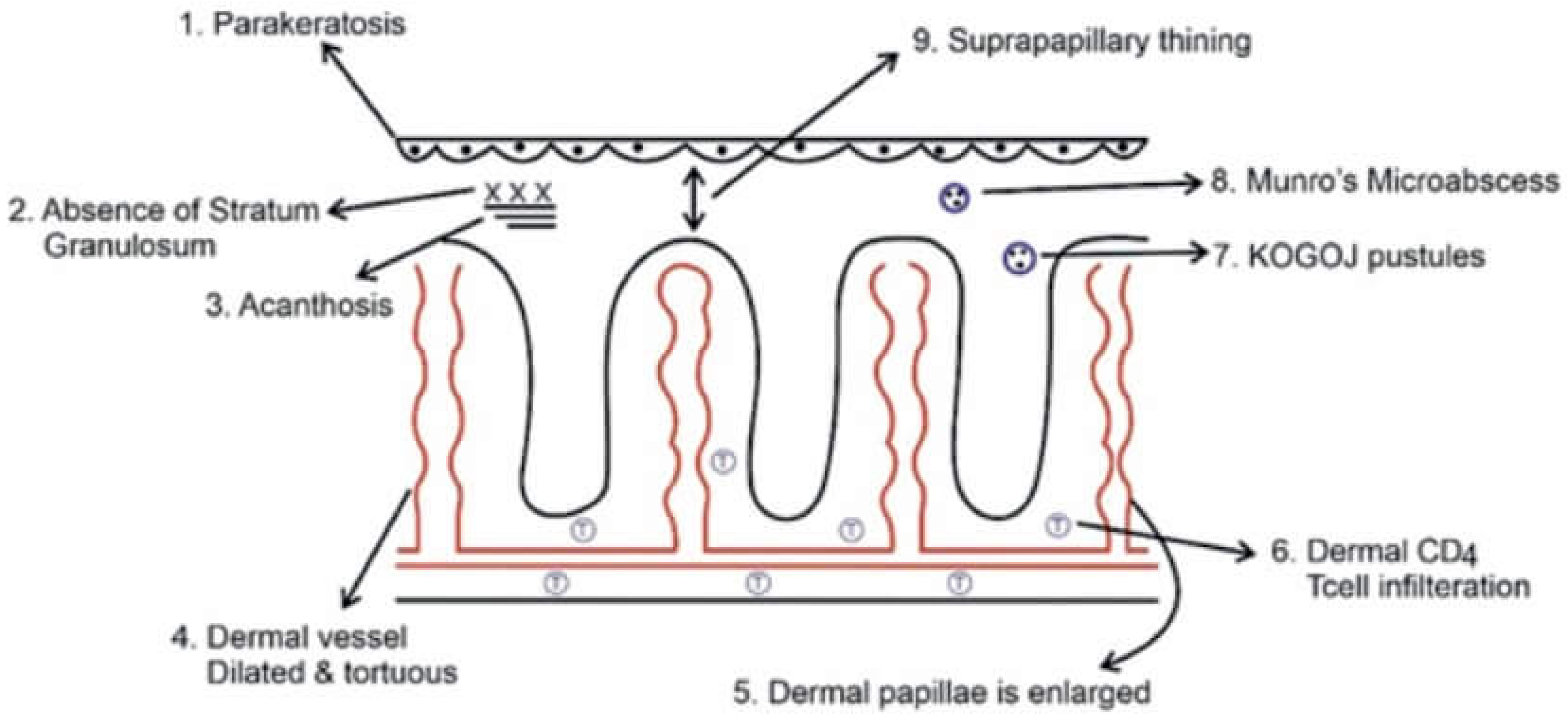
1. Grattage Test – Increased accentuation of scales
2. Candle grease sign – when final layer of scale come out.



wornoff ring- surrounded hypopigmentd margin(It is due to prostagladnins secreted)
&
central shinning- **membrane burkeley membrane**
(usually seen after phototherapy)

3. Auspitz sign→ pin point bleeding spots.

Histology-


1. Parakeratosis
9. Suprapapillary thining
2. Absence of Stratum Granulosum
8. Munro's Microabscess
7. KOGOJ pustules
3. Acanthosis
6. Dermal CD4 Tcell infilteration
4. Dermal vessel Dilated & tortuous
5. Dermal papillae is enlarged

1. Parakeratosis (retention of nucleus in topmost layer)
2. absence of stratum granulosum
3. Acanthosis (increases thickness of St. spinosum)
4. Dermal vessel is dilated and tortuous. Dermal papillae is enlarged.
6. (due to) dermal CD4 Tcell infiltration
7. Kogoj pustules (collection of neutrophilic)
8. Munro's microabscess
9. Suprapillary thining

→ **Severe Types of Psoriasis.**

- There are complicated psoriasis like-

1) Pustular psoriasis

→ Multiple sterile pustule develops on palm and sole (as no infection only infiltration present)

→ If generalized k/a Von Zumbusch disease

→ If patient pregnant has disease k/a Impetigo herpetiformis

2) Erythrodermic psoriasis

→ Psoriasis with Inflamed / red lesions involving whole skin

→ Both these complications develop on withdrawal of systemic corticosteroid

∴ They are C/I in Psoriasis

→ Psoriasis is an inflammatory condition. In any inflammatory Condition, administration of corticosteroid will subside inflammation but on tapering of steroids rebound phenomenon occurs where more severe form of psoriasis develop.

Complication:

1. Psoriatic arthritis

Different Types:

- i. Classic – DIP Type - DIP Involvement
- ii. Asymmetrical oligo articular type
- iii. symmetrical poly articular type

iv. Arthritis mutilans
v. Exoskeleton involvement
type i.e.- Spondylitis/ sacroiliac joint involvement

Q. MC joint involved in Psoriatic arthritis?
Ans. Dip

Q. MC type of psoriatic arthritis?
Ans. Asymmetrical oligo articular type.

Treatment

A. Topical

1. Corticosteroid (systemic Corticosteroid are C/I)
2. Keratolytic agents (helps remove scaling)
    - i) Salicylic acid Q DNB' 17
    - ii) Coal tar
3. Vitamin D analogs
    - i) Calcitriol
    - ii) Calcipotriol
4. Topical retinoid - Tazarotene

B. Systemic

1. Methotrexate
S/e: Hepatotoxic, Teratogenic
2. Cyclosporine
S/e: Nephrotoxic, HTN, Hypertriglyceridemia, Hypertrichosis
3. Acitretin (systemic retinoid)
S/e: Teratogenic, Dyslipidemia

C. **Phototherapy**PUVA, PUVA Sol. NBUVB, Target phototherapy

Q **Specifically for Psoriasis**
Ingram regimen → Dithranol application → expose to light
Gaekerman regimen→ Coal tar application→ expose to light

D. Biological

Monoclonal Antibodies

Infliximab
Adalimumab
Etanercept
} TNF α Inhibitor

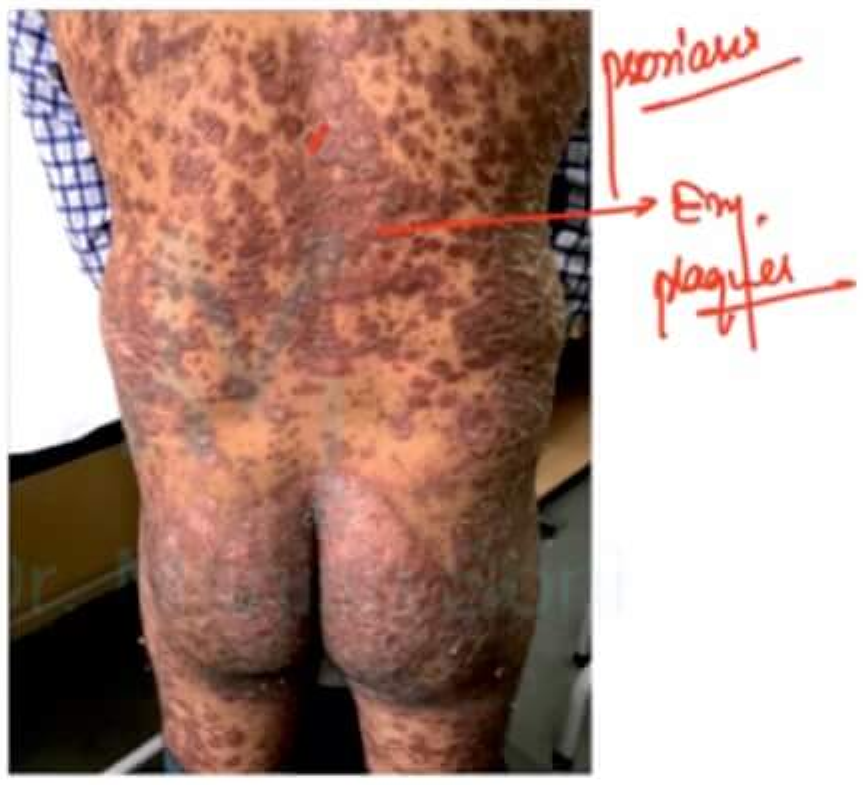
Efalizumab → bind to CD-11
Itolizumab → Bind to CD-6
Alefacept → bind to CD-2
} T - Cell Inhibitor

Apremilast → PDE- 4 inhibitor
Secukinumab → anti IL – 17A
JIPMER 17 Q ustekinumab → Anti IL – 12/23

Important fact

Doc for:

1. Psoriatic arthritis → Methotrexate
2. Erythrodermic psoriasis → Methotrexate
3. Pustular Psoriasis → Acitretin > Methotrexate Q
4. Impetigo Herpetiformis → Systemic corticosteroid (pregnancy B Category) > Cyclosporine (pregnancy c category)



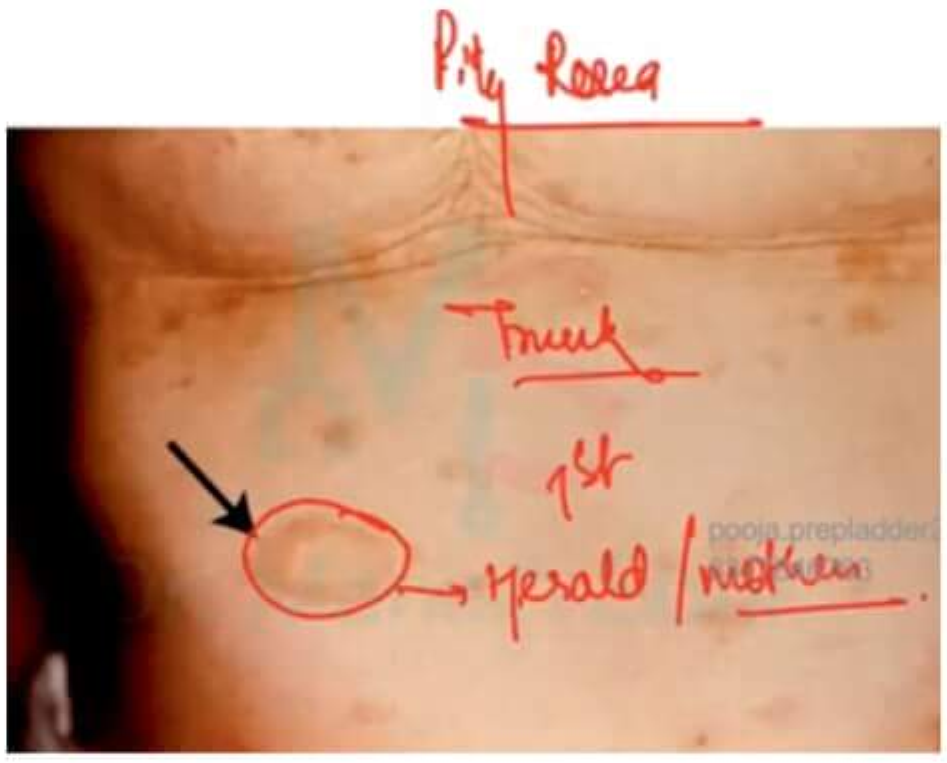
2. Pityriasis Rosecea

↓ Means ↓
Scaling Pink/red

- This disorder is characterized by pink/red lessons with scaling

"Her	trunk	and	cigarette	on	fire"
↓	↓		↓	↓	↓
Her>His	M.C site		(Cigarette Paper scaling or Collarette of scales)	Itself (self-limiting)	Fir tree appearance
↓					
Herald Patch/mother Patch					

Associated with Herpes (Human Herpes Virus 7,6)



3. Lichen planus.

→ It is a autoimmune disorder

→ Usually clinically involves all the sites of body, skin, mucosa, nail or hair.

C/F

I. Skin- 5 P's

→ Plain, purple, polygonal, pruritic papule

→ White reticulate pattern K/a Wickham's striae

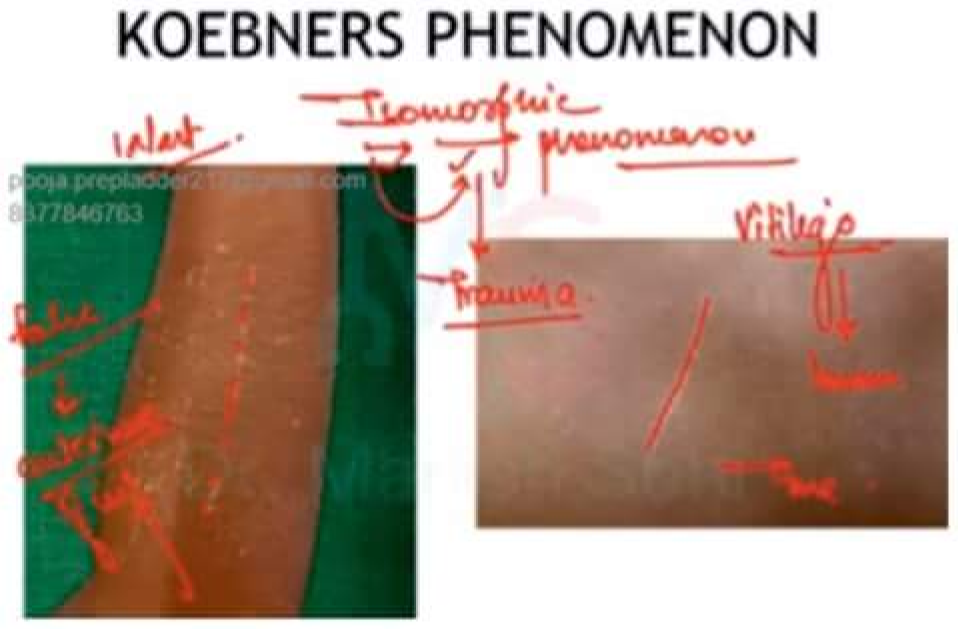
↓

(are due to wedge shaped hypergranulosis& thickening of stratum Granulosum)

→ Develop commonly over flexors

→ Sometimes associated with liver disorders like Hep C, Hep B, Primary biliary cirrhosis

→ These lesions start developing along the lines or trauma k/a isomorphic phenomenon / koebners phenomenon (i.e. same type of lesion develops a long line of trauma)



Koebners phenomenon can also be associated with

1. **True koebner phenomenon** – Psoriasis, lichen planus,vitiligo
2. **False koebner phenomenon** – Viral warts, molluscum contagiosum

→ Here Since it is an infective condition and this phenomenon is seen due to auto-inoculation, so infection spreads through nail to different sites.

3. **Occasional koebner phen**omenon – Kaposi sarcoma
   – Lichen nitidus
   – Lichen sclerosis

Other rare causes-

4. **Reverse koebner phen**omenon. Psoriasis, granuloma annulare
5. **Remote reverse koebner phen**omenon – vitiligo

II Mucosa-

→ Generally, oral mucosa > genital mucosal involvement is seen.

- **Oral Mucosa**- Shows white lacy pattern or white reticulate pattern, sometimes lesions are ulcerative or erosive which may progress to squamous cell carcinoma
  [but lichen planus is not premalignant condition]

- **Genital Mucosa**- Shows annular lesions

III. Nails-

1. Most characteristic nail change is pterygium (that is wing shaped projection of proximal nail fold)
2. Late in disease: Anonychia: absence of nail
3. Onychorexhia→ brittle nails with longitudinaL plate ridging.
4. Trachyonychia → sand paper like rough nails(all 20 nails get involved are dystrophied)

20 nail dystrophy can also be associated with

i) P- Psoriasis
ii) A- Alopecia areata
iii) L- Lichen planus
iv) E- Eczema

IV. Hair-

→ Lichen planus with hair changes involvement is k/a lichen plano pilaris/ follicular lichen planus
→ Initially it presents with follicular hyperpigmented lesions over scalps which heal with scarring alopecia

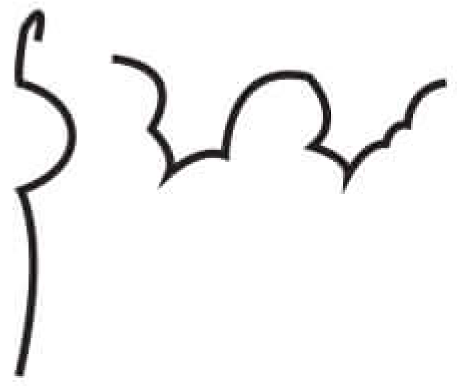
Types of lichen planus based on type of lesion

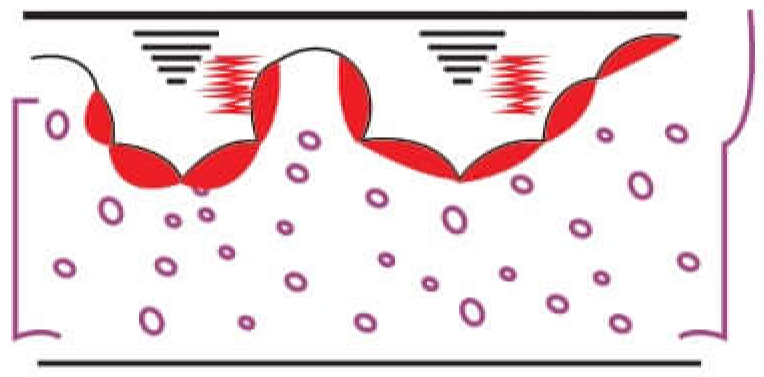
1. Annular lichen planus
2. Linear lichen planus
3. Hypertrophic lichen planus
4. Actinic lichen planus (occurring over sun espoused area)
5. Lichen planus pigmentosus
6. Lichen planus pilaris
7. Lichen planus pemphigoides (shows bullous lesions)

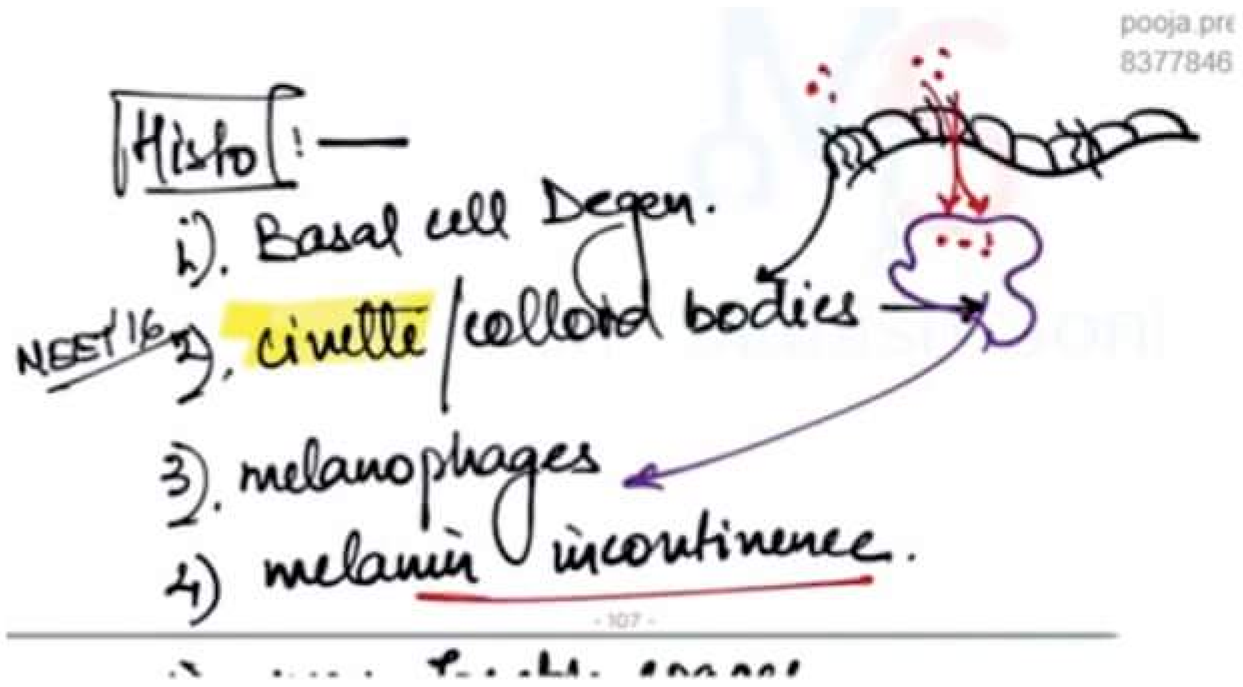
Investigations

1. **Histology:**

a. Basal cell degenerations
b. Civette^{Q}/ colloid bodies – There are degenerating basal keratinocytes.
c. Melanophages- Melanin containing macrophages
d. melanin incontinence {i.e leaking of melanin from epidermis to dermis}
e. Max joseph spaces
f. Saw toothed appearance
g. Hypergranulosis (wedge shaped)
h. Band like lymphocytic infiltrate in upper dermis
I. Irregular Acanthosis
j. Interface dermatitis (i.e. basal cell degeneration in lower epidermis and band like infiltrate in upper dermis)


Saw toothed appearance


max joseph spaces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Histo :—

1). Basal cell Degen.

NEET 16 2). civette/colloid bodies

3). melanophages

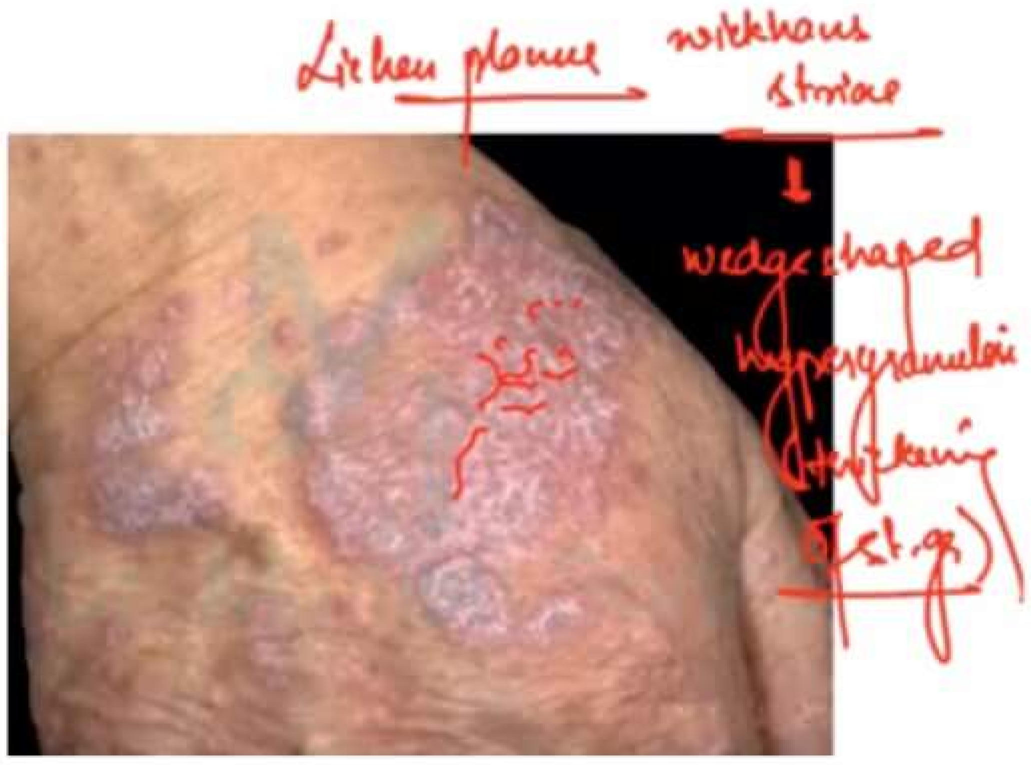
4) melanin incontinence.

Treatment

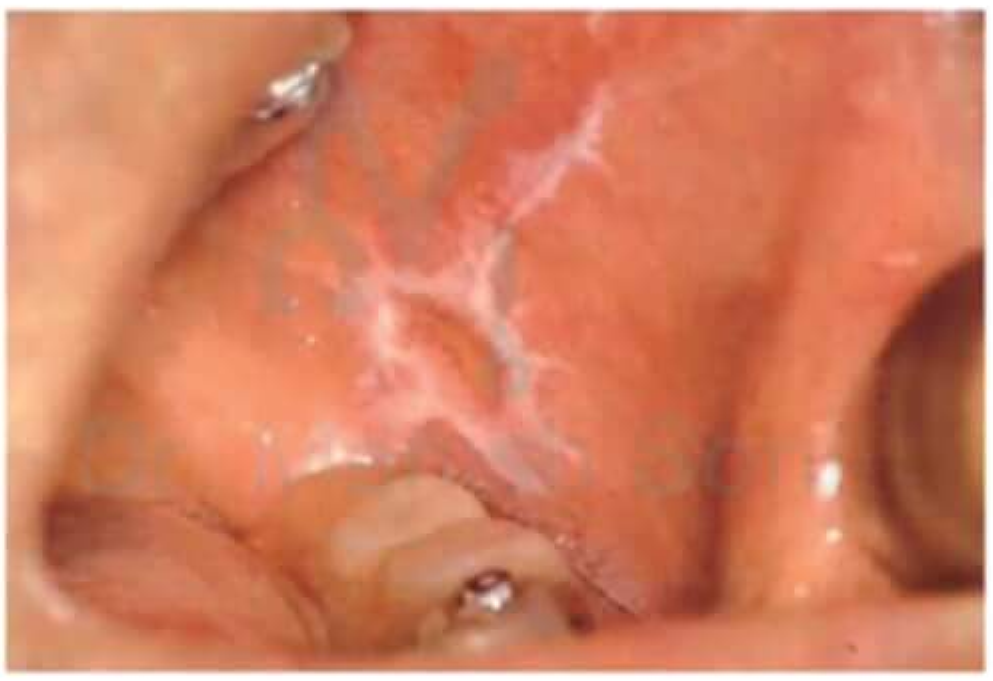
→ Steroid – mainstay of Rx
- Topical
- Systemic
- Intralesional

Other azathioprine
Dapsone

→ In generalized cases phototherapy given

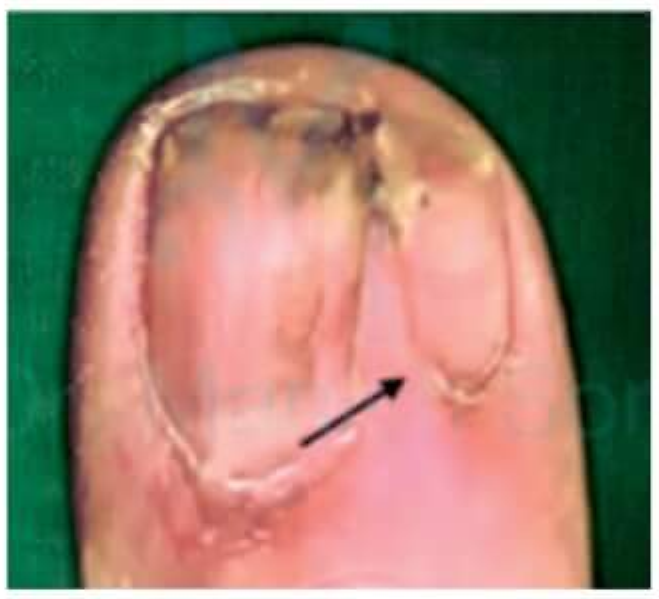


LICHEN PLANUS WITH AMULGUM

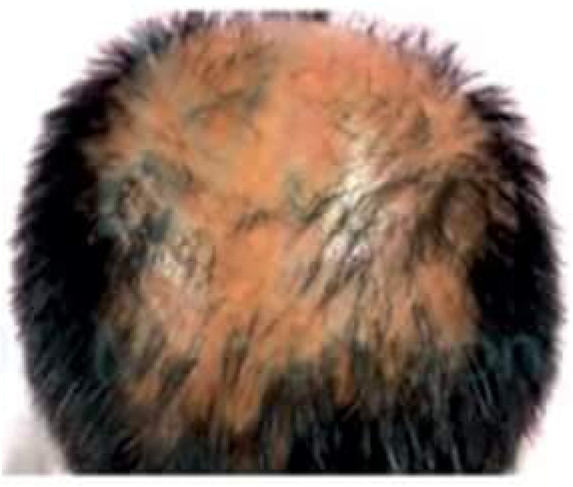


lichen planus is associated with amalgam/mercury (used in dental fillings)

PTERYGIUM



LICHEN PLANO PILARIS

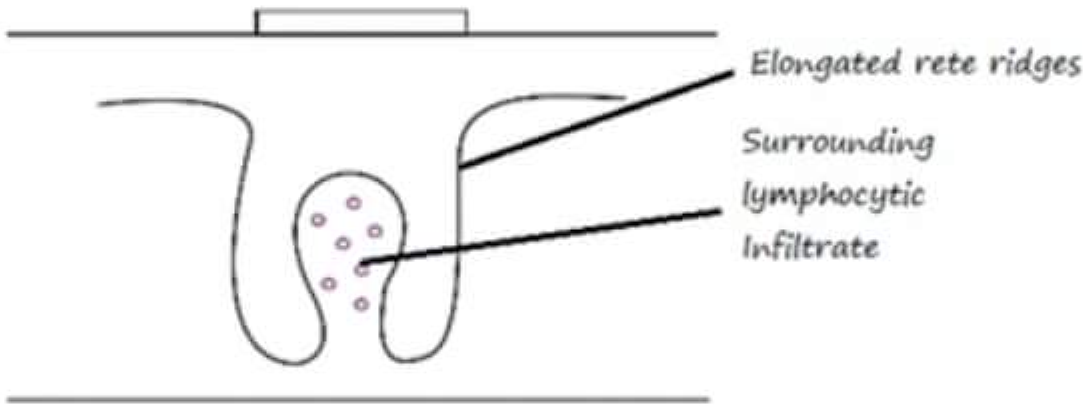


4. Lichen Nitidus

→ Clinically present with tiny papules over dorsum of hand, forearm, elbow, shaft of penis.

Investigation

1. Histology



Giving a claw clutching appearance

Treatment

- Topical - Corticosteroid
  If thickening of lesion add salicylic acid (keratolytic agent)
- Systemic - Acitretin

5. Pityriasis rubra pilaris

Pityriasis	rubra	pilaris
↓	↓	↓
Scaling	red	hair/ follicular

→ Clinically present with multiple follicular scaly prominences with orange-red hue

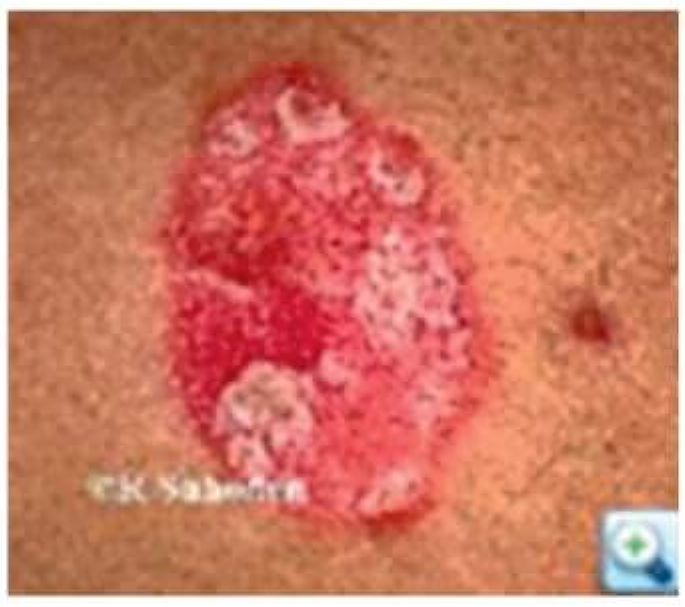
→ Over trunk with areas of normal / healthy skin in between k/a islands **of normal skin.**

→ Griffith's classification:

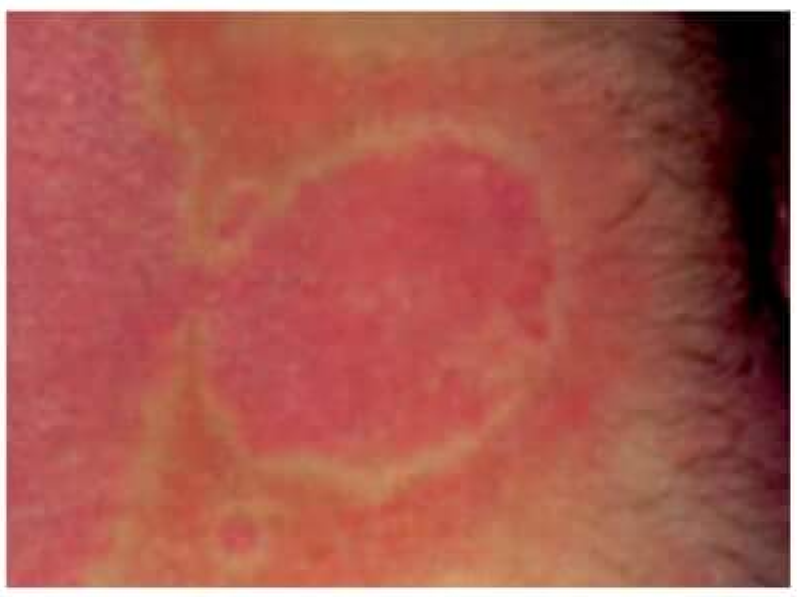
- Divides it in 6 types
- Type 6 is associated with HIV

→ **Treatment**

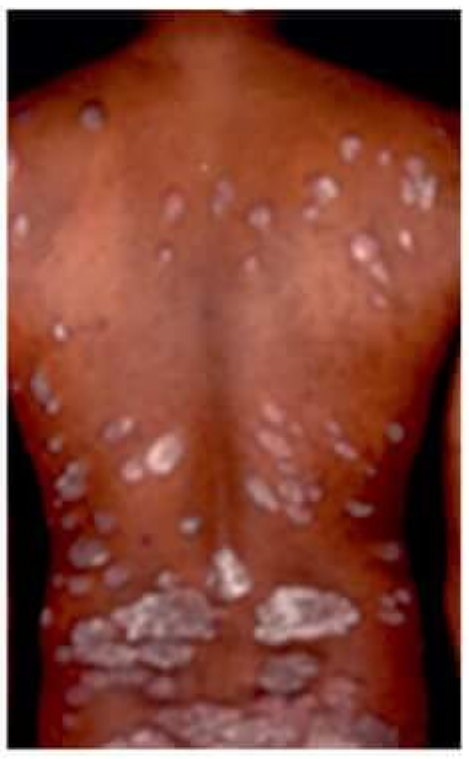
- Topical : Corticosteroid + Salicylic acid (if thickening)
- Systemic: Acitretin



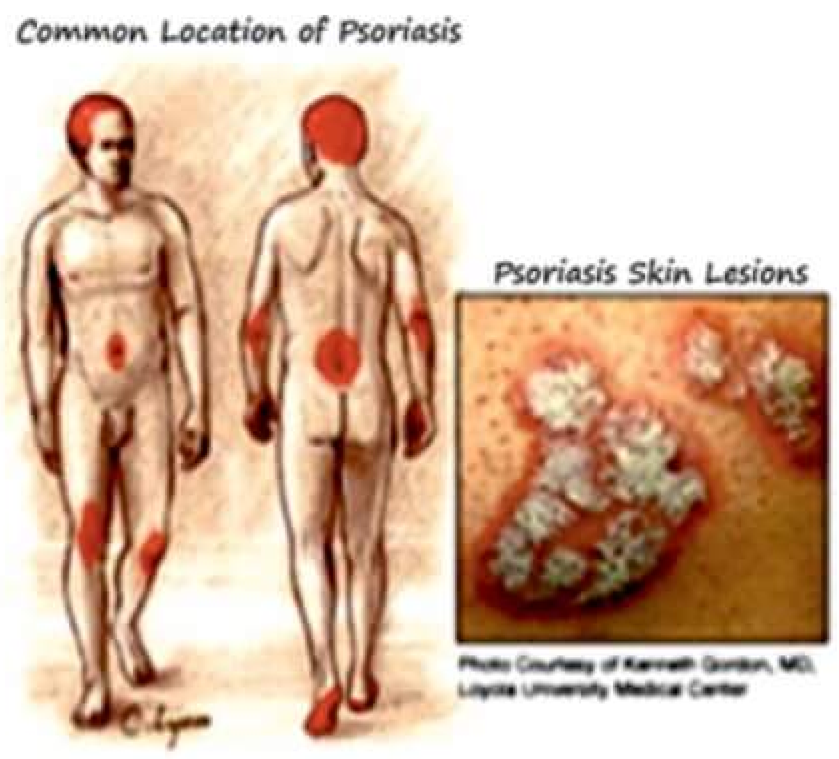
Auspitz's sign

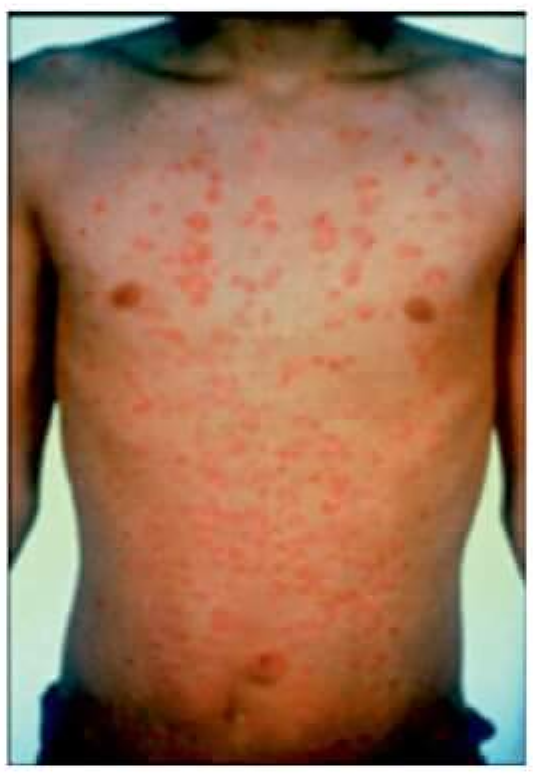


wornonoff ring

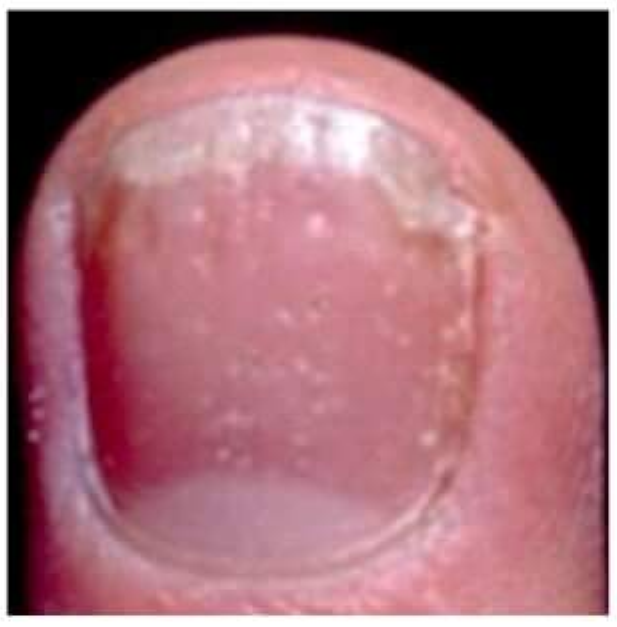


plaque psoriasis

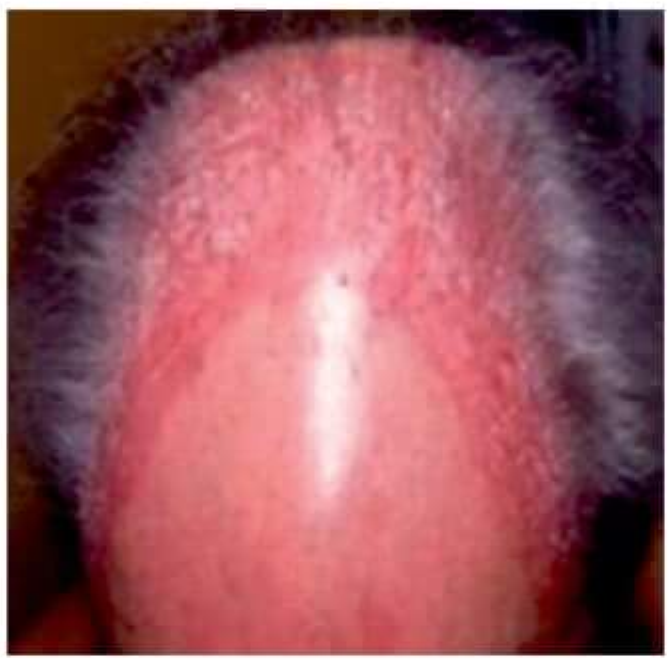

Common Location of Psoriasis
Psoriasis Skin Lesions
Photo Courtesy of Kenneth Gordon, MD, Loyola University Medical Center



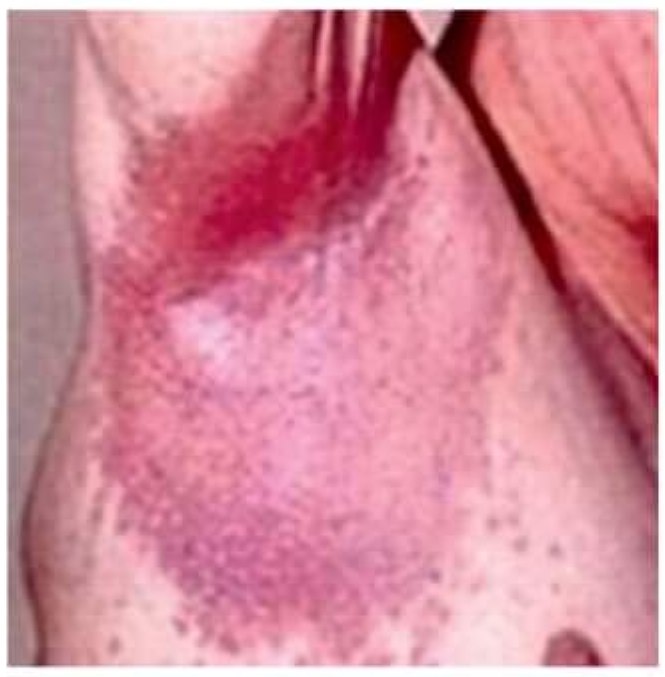
Guttate psoriasis



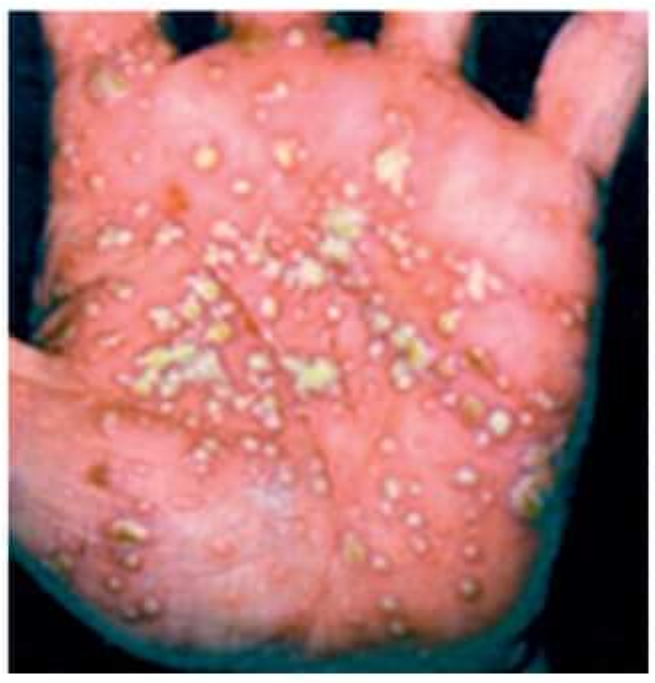
nail psoriasis



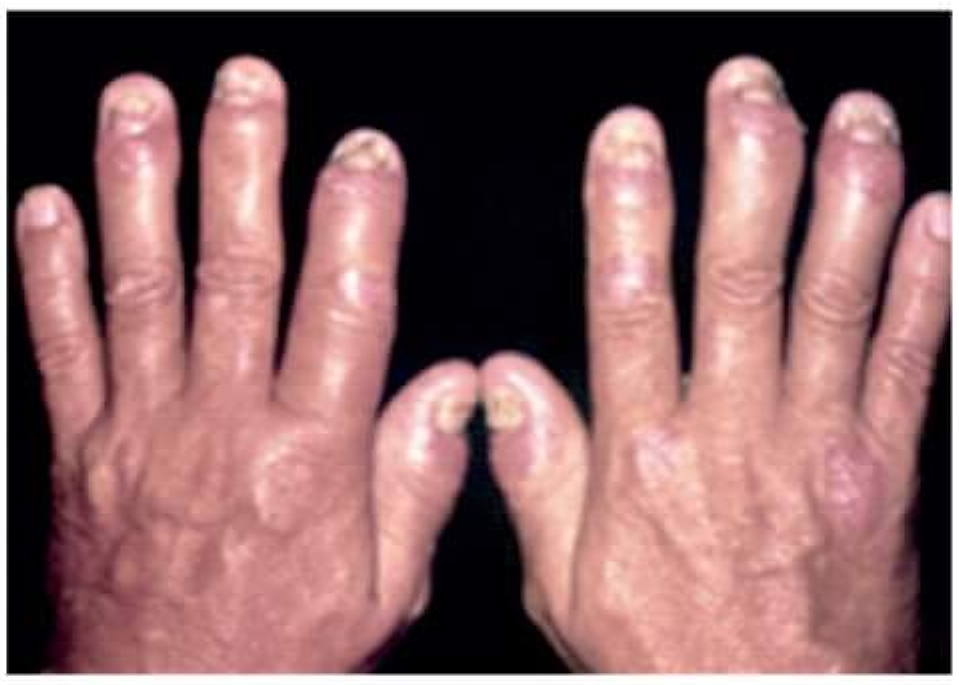
Scalp psoriasis



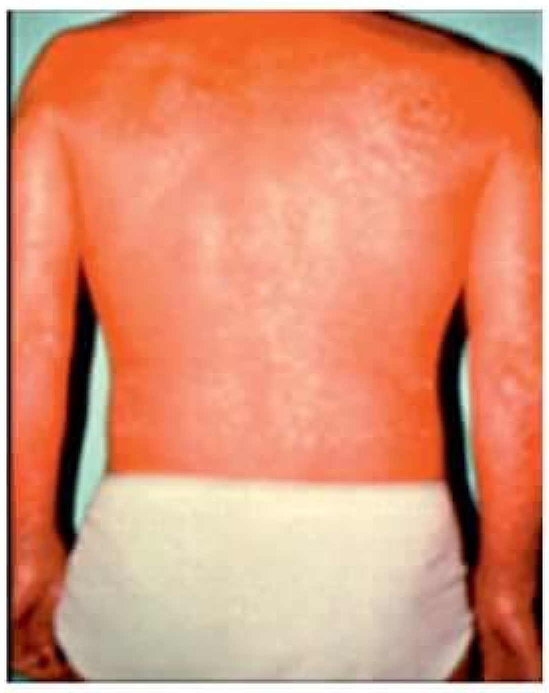
inverse psoriasis



pustular psoriasis

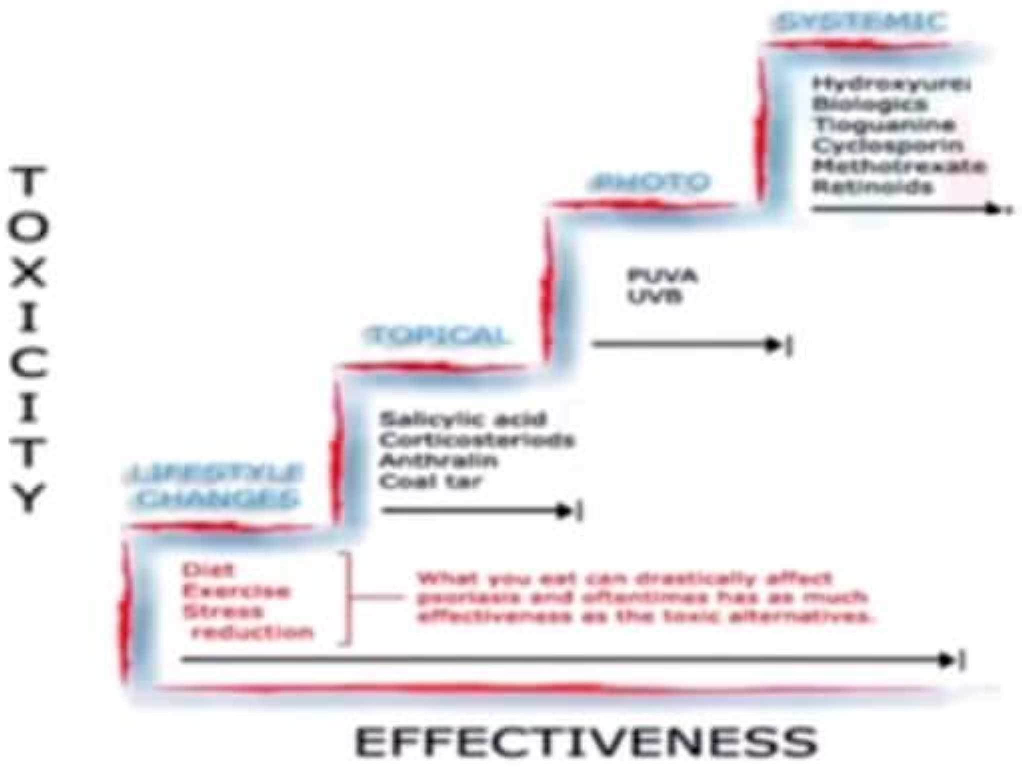


Psoriatic arthritis



erythrodermic psoriasis

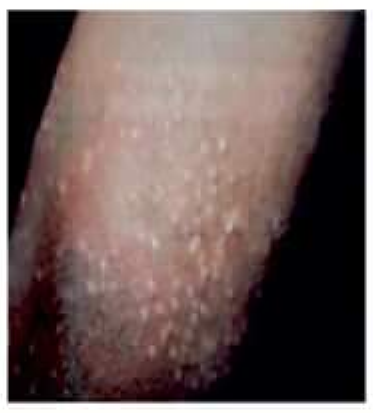
Treatment ladder

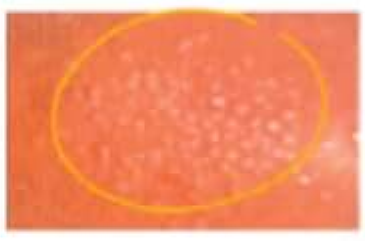


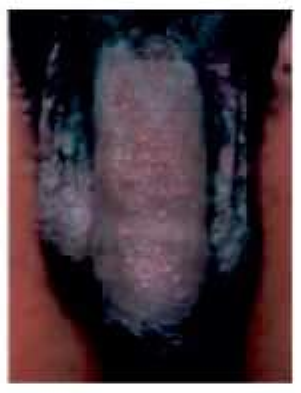
Q. A child came with similar lesions over elbows, shaft or penis and forearm. Shows pinhead discrete but grouped papules over right elbow. Diagnosis?

A. Lichen nitidus
B. Lichen planus
C. Scabies
D. Phrynoderma

Ans- A

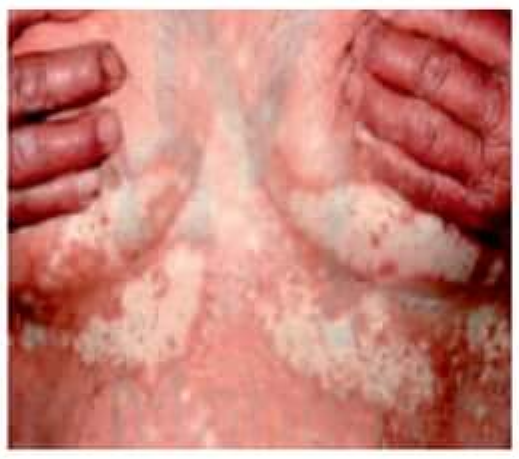






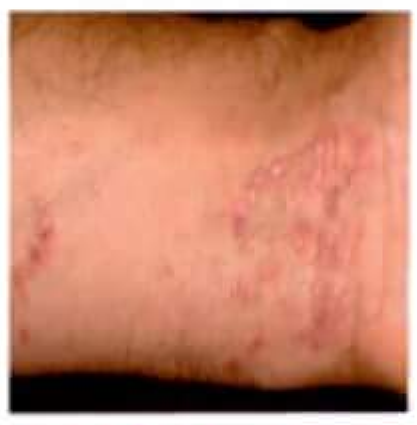
Q. An eruption of follicular hyperkeratotic papules of reddish orange color spreading in a cephalocaudal direction. Conflunece to psoriasiform, scaling dermatitis with sharply demarcated islands of unaffected skin. With progression to erythroderma. Which of the following classification is used for above mentioned disease?

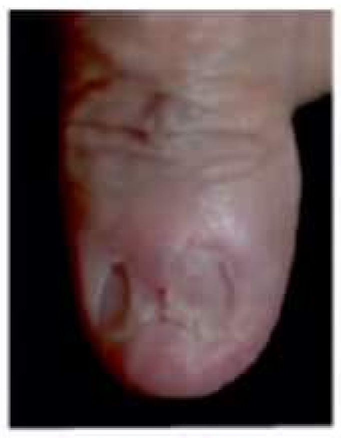
A. Lopez classification
B. Mark rubin classification
C. Gonzales classification
D. Griffiths classification

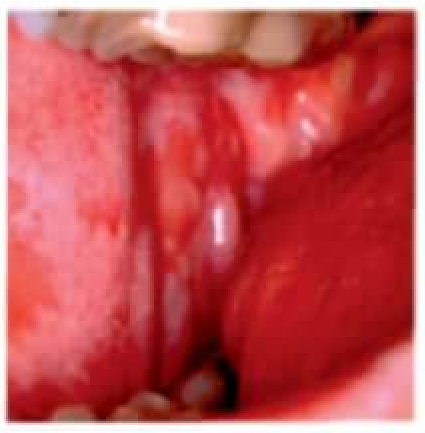


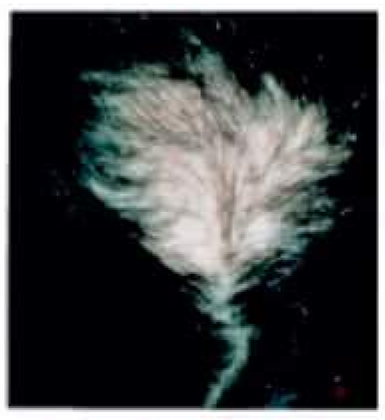
Ans-D

Feature of Lichen planus


5P's of Lichen Planus

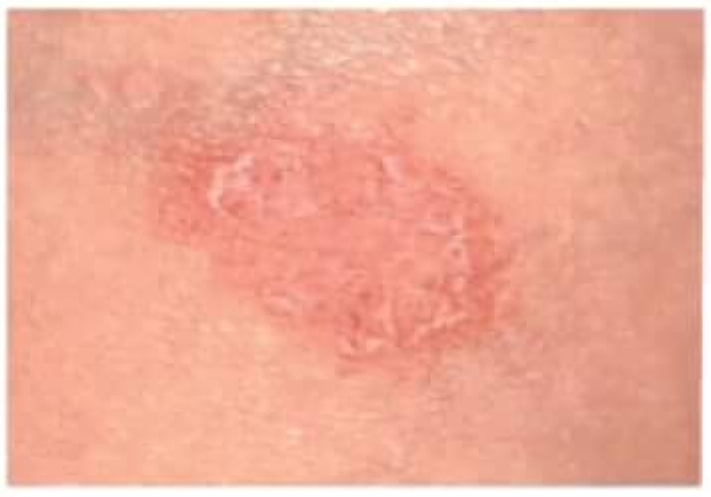

Eponychium

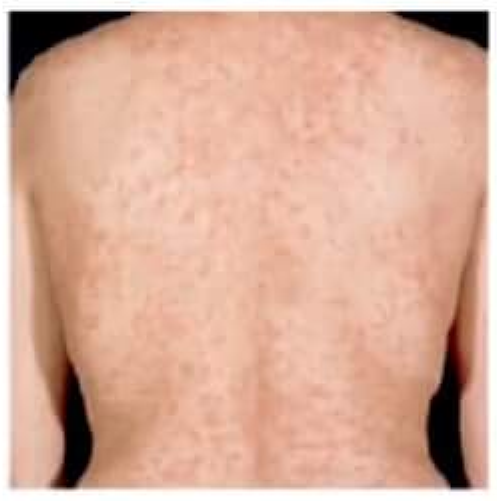

White lacy pattern over buccal mucosa


Scarring alopecia

5P's of Lichen planus, Pterygium, white lacy pattern over buccal mucosa, scarring alopecia

Features seen in Pityriasis rosacea


Hanging Curtain Sign


Fur tree like appearance of lesions

MISCELLANEOUS DISORDERS

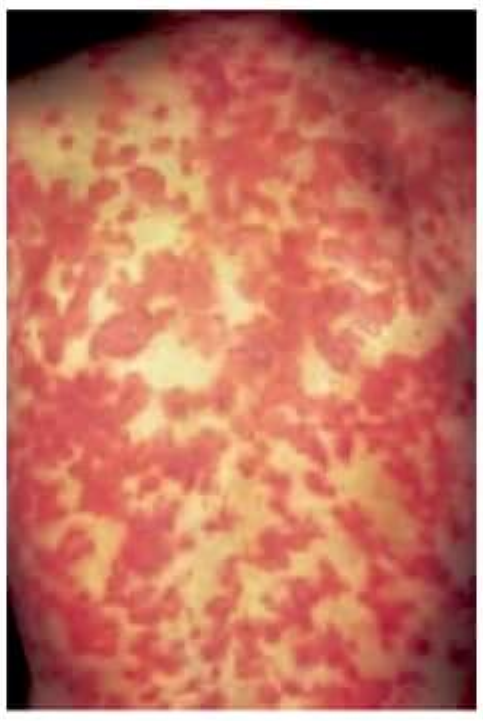
1. MYCOSIS FUNGODIES / CUTANEOUS T- CELL LYMPHOMA

3 Clinical Stages:

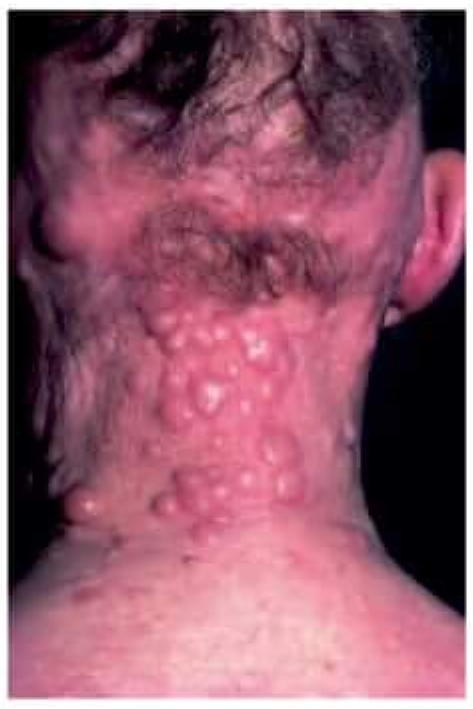
Stage Name	Treatment
1. Plaque	Full skin electron beam therapy
2. Tumor	Chemotherapy
3. Patch	Topical nitrogen mustard

Histology

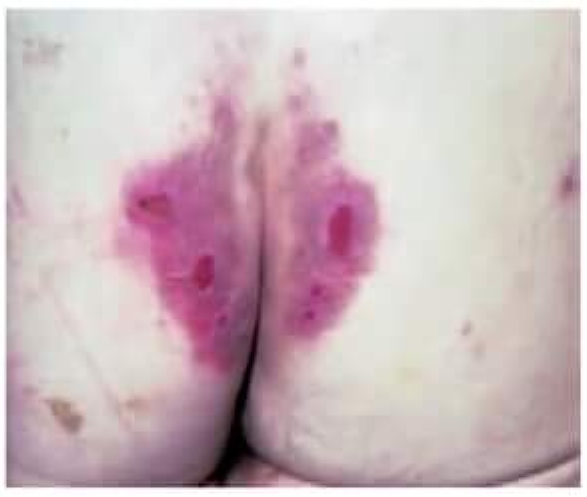
1. **Sezary cell:** malignant or atypical lymphocytes with cerebriform nucleus
2. Epidermotropism
3. Pautrier's micro abscess



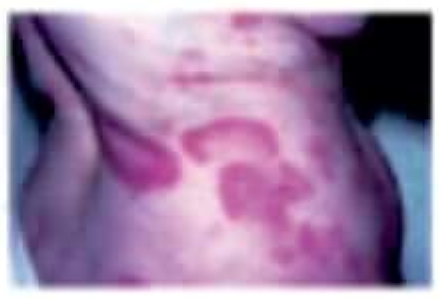
Mycosis fungoides plaques with extensive involvement



nodular Mycosis fungoides showing nodules on the back of the neck



early Mycosis fungoides patch



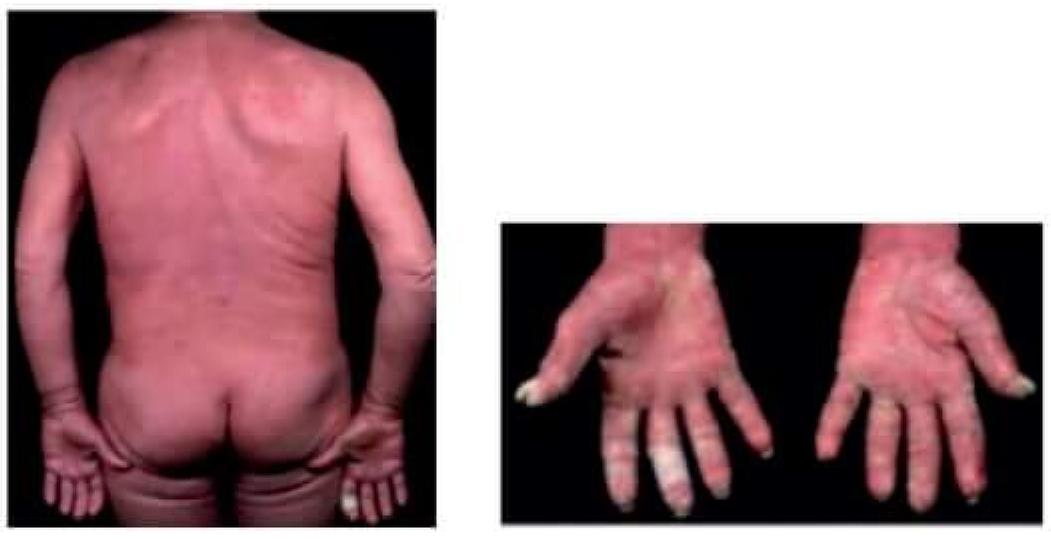
advanced Mycosis fungoides plaques

2. SEZARY SYNDROME

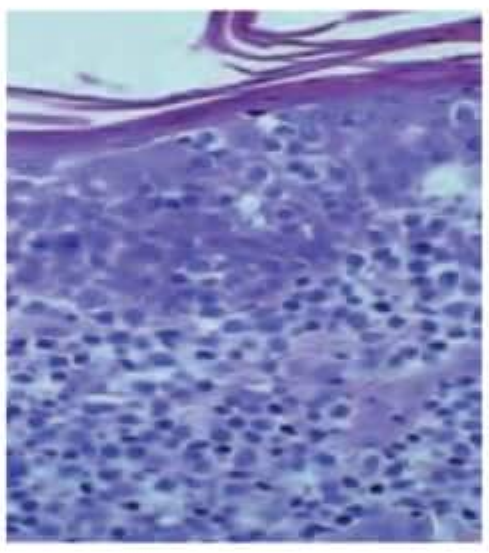
Leukemic / Erythrodermic variant of mycosis fungoides

3 Characteristics:

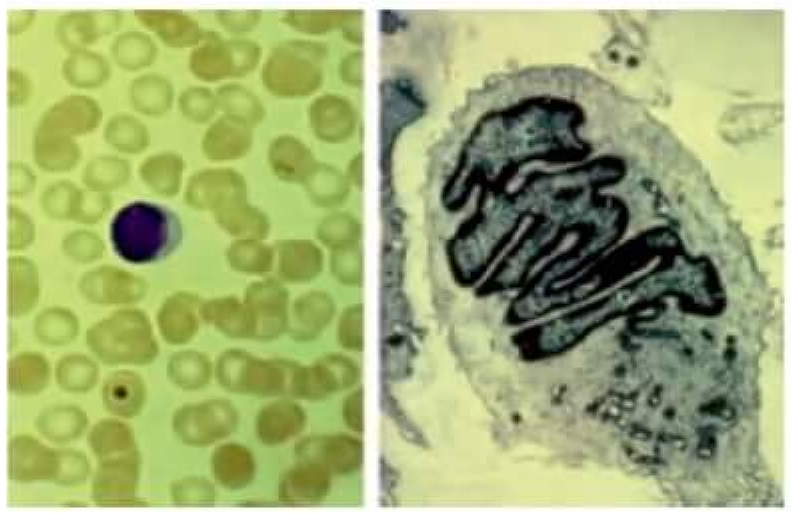
i)S - Sezary cell in peripheral blood
ii)L -Lymphadenopathy
iii)E -Erythroderma with palmo plantar hyperkeratosis.



Erythroderma with palmoplantar hyperkeratosis



Showing epidermotropism with the presence of cytologically atypical small dark cells



Sezary cells in peripheral blood showing features of atypical cerebriform nucleus.

3. NEUTROPHILIC DERMATOSIS -deep dermal neutrophilic infiltrate is seen

Sweet syndrome or Acute febrile neutrophilic dermatosis	pyoderma gangrenosum
1) Tender erythematous nodule with fever, increased ESR and leukocytosis	1) Ulcers with undermined edges.
2) Associated with AML, Adenocarcinoma.	2) Associated with IBD(UC>CD) RA, SLE, CML, NHL
3) Drug of choice: Systemic Corticosteroid	3) Drug of choice: Systemic Corticosteroids

4) ICHTHYOSIS

Characterized by- → Severe dryness
→ Fish like scales

3 Common type		
Ichthyosis Vulgaris (MC)	X-linked Ichthyosis	Lamellar ichthyosis
Autosomal dominant	X-linked recessive (XLR)	Autosomal recessive
Deficiency of filaggrin	Deficiency of steroid sulphatase enzyme	Deficiency of transglutaminase -1 enzyme
Extensors mainly involved	Extensors and flexors involved	Whole body involved ↓ Colloidion membrane / Baby

Harlequin Ichthyosis

- → Autosomal recessive
- → Gene (A B C A – 12) defect
- → Most severe

5)Parasitology and Infestations

i) Migratory infection of skin

Cutaneous Larva migrans	Larva currens	Calabar swelling
-caused by filariform larva of animal (not human) hookworm	- Strongyloides stercoralis	-Loa Loa
-C/F serpiginous trunk	-C/F urticarial rash	-C/F painless nonpitting Non erythematous Swelling or edema
Treatment-Ivermectin+ Albendazole	Ivermectin	DEC (Diethyl carbamazine)

ii) Trypanosomiasis

- → Winter bottom sign- posterior cervical Lymphadenopathy – seen in early phase of African trypanosomiasis
- → Romana sign -Unilateral, periorbital, palpable painless edema. – seen in Acute phase of American trypanosomiasis

iii) Leishmaniasis

Cutaneous

Old world
Leishmania Major
-delhi boil
Ulcer with Central crusting

New world
Leishmania Mexicana
-Chiclero ulcer(Gum tree harvestors's ulcer)

Mucocutaneous

-skin mucosa involved.
- mutilation present
Called as Espundia

Visceral

- Kala azar
if inadequately treated
Progresses to - PKDL
(Post kala azar dermal Leishmaniasis)
i) hypopigmented lesions over trunk
ii) nodular lesions over face
iii)Endemic areas : Bihar

On Giemsa stain: LD bodies -Leishman Donovan bodies Seen in leishmaniasis.

4 Various types of infestations:

A. Pediculosis – Infestations with lice

Head	Body	Pubic
↓	↓	↓
Pediculosis capitis ↓	Pediculosis Corporis / Vagabond disease	Pthysis Pubis
Clinical feature → – 'Girl' associated With regional Lymphadenopathy – Nits (egg cases) Attached to hair Follicles	→ Itching ↓ (ERRORUM MORBUS)	→ Maculae Cerulea ↓ Sexually transmitted infection
Treatment → Application of Permethrine (1%)	1. **Disinfection of clothes**^{Q} 2. Application of Permethrin (1%)	1. Shaving of pubic hair 2. Application of Permethrin (1%) 3. Treatment of contact

2) Scabies →

Caused by: - Sarcoptes scabies var hominis
- Incubation is 4 weeks
- If patient re infested with mites → ICP is 2 days

Clinical features:

I. Burrow's
→ Most characteristic
→ Linear also known as 'Serpiginous, therefore, they appear S Shape'
→ Formed in Stratum corneum

II. Papules, nodules, vesicles – Hypersensitivity to mites
Nodular – Seen in scrotum
↓
(Nodular / Genital Scabies)
Vesicular → Palm and sole in infants

Diagnosis:
- → Itchy (Nocturnal itchy)
- → Presence of burrow
- → Family history positive
- → Distribution of lesions

Distribution of lesions

Face, Palm, Soles
→ Adults (Spared)
→ Infants (Involved)

Circle of Hebra
→ Web space
→ Groin
→ Periumbilical area
→ Flexors of writs, elbow & axilla

To confirm diagnosis:
Prepare 10% KOH - i) identify mite
ii) eggs
iii) fecal pellets (scybala)

Most severe type → Norwegian / Crusted Scabies

- Treatment:

Topical agents
1) Permethrin 5% DOC single application (Acts on sodium channel)

2) GBHC (Gamma Benzene Hexachloride) 1% (C/I in pregnancy, children < 2yrs, h/O epilepsy because it is a neurotoxin.)
3) Benzyl benzoate 25% (disadvantage - multiple applications required)
4) Crotamiton 10% -Antipruritic
5) Precipitated Sulphur

Systemic agent:

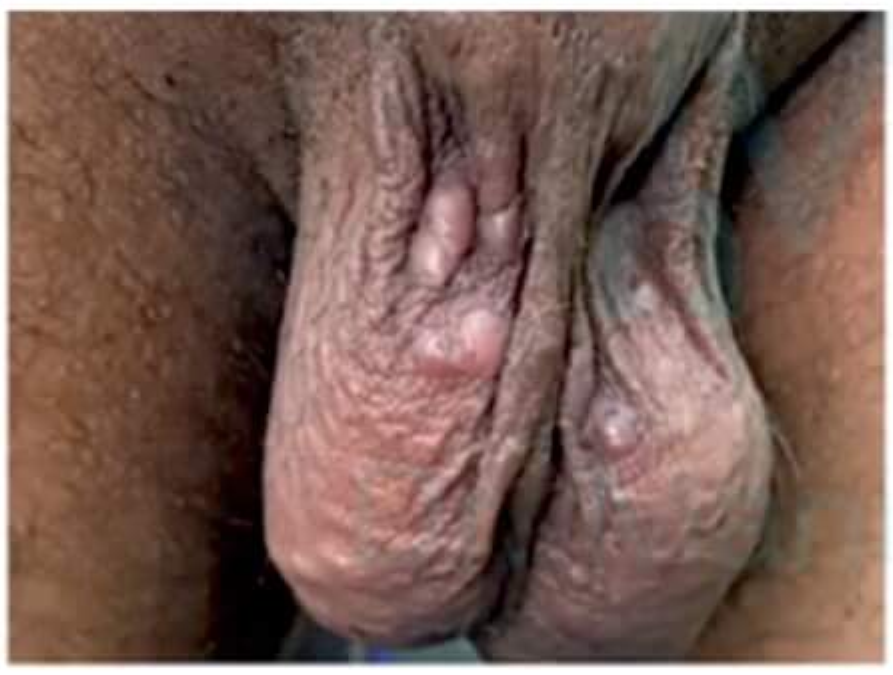
1)Ivermectin -0.2 mg/kg/Bwt (200 microgram/kg/Bwt)

(Acts on glutamate gated chloride channels – Hyperpolarisation [chloride influx]
Leads to paralysis of mite)

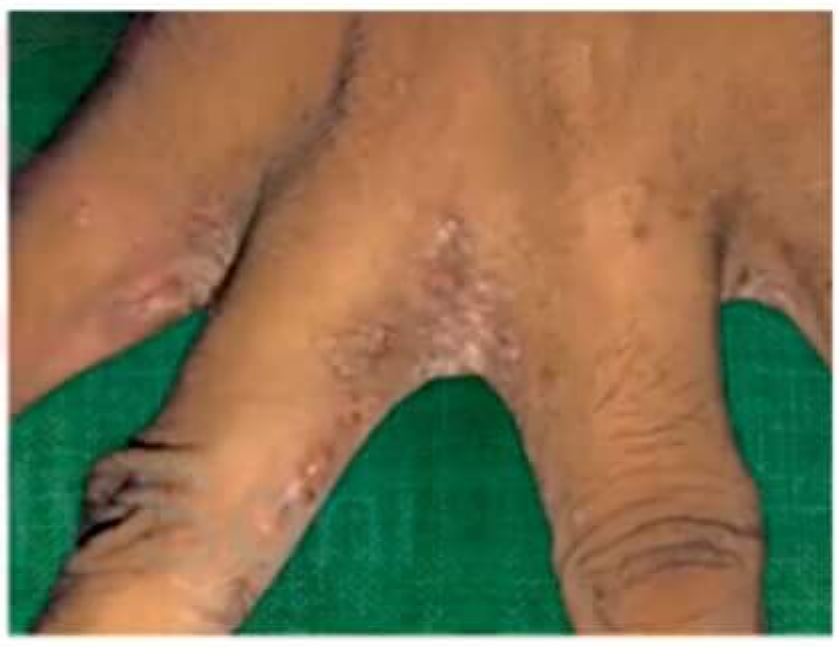
→ Most severe scabies- Norwegian scabies / crusted scabies)
↓
Millions of mite
↓
Pruritus

→ Seen in old patients, bed ridden patients, psychiatric patients,
Leprosy patients with decreased sensation.

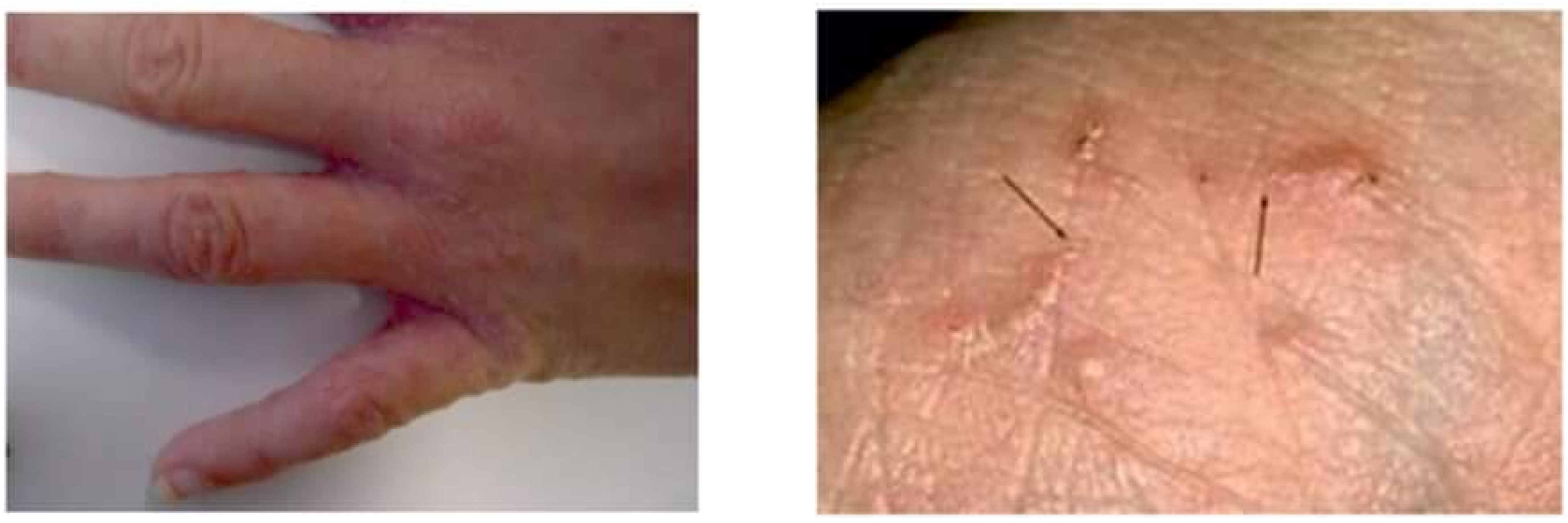
→ Ivermectin atleast 2 doses along with Permethrin multiple applications should be taken after 2 weeks.



Nodular lesions over scrotum



Lesions present on web spaces



Burrows seen in the above image are formed in stratum corneum

3) Dermatitis / Eczema

↓ ↓

Exogenous Endogenous

Exogenous Eczema:

1. Irritant contact dermatitis

Due to detergents (Most common) and vegetable
Seen in housewives, that's why called as housewives' eczema

2. **Allergic contact dermatitis**
   - Nickel (Most common) → **Ornaments**
   - Potassium dichromate → **cement** Q
   - Para phenylene diamine **(Hair dye)**

   Diagnosis: Patch test

   → Lesion do not develop on 1^{st} exposure.

   → Initially Memory Tcells are formed and then patients develop dermatitis after multiple exposure.

3. **Air borne contact dermatitis**

   - occurs by congress grass → **sesquesterpine lactone (causative allergen) / parthenium hysterophorus**

   Patch test

   → Patient positive for sequesterpine lactone

Endogenous Eczema:

1. Nummular / Discoid – coin
2. Pompholyx / Dyshidrotic: Sago grain / tapioca feel
3. Asteatotic / winter due to cold
4. Atopic Dermatitis – increase trans epidermal water loss Q

↓

Clinical feature: Dry / Xerosis skin

↓

Itching

↓

Scratching

Itch – scratch – itch cycle

-Cradle crap / seborrheic dermatitis: infants

Diagnosis: Hanifin and Rajka Criteria

4 major

→ Pruritus

→ Chronic

→ Family history of Atopy

→ Distribution of lesion

- Adult ↓ Flexors
- Child ↓ Flexors, Face
- Infants ↓ Flexors, Face, Extensors

23 minor

Few Minor Criteria:

a) Dennie Morgan fold – extra skin fold in lower eyelid.

b) White dermographism

c) Allergic conjunctivitis

d) P. alba

e) ASSC

f) Keratoconus

g) wool intolerance

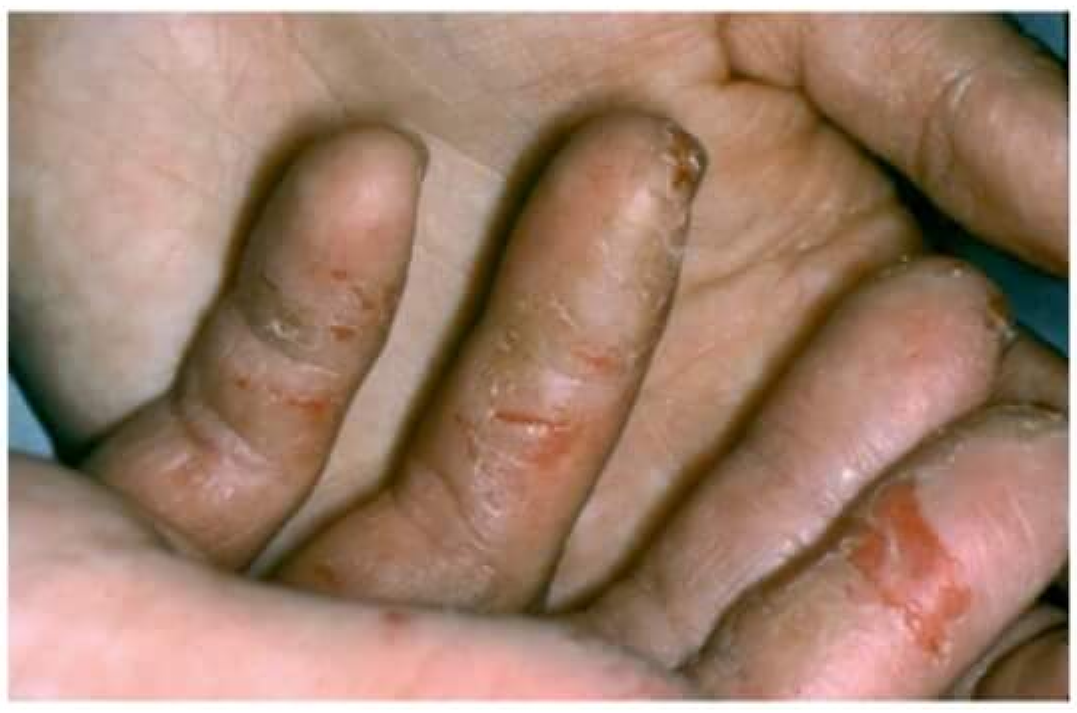
h) Food intolerance

NEET 2018 Q – child, itchy, lichenified lesions over flexor of elbow and knee
H/o Asthma in parents. diagnosis?
Answer- Atopic dermatitis

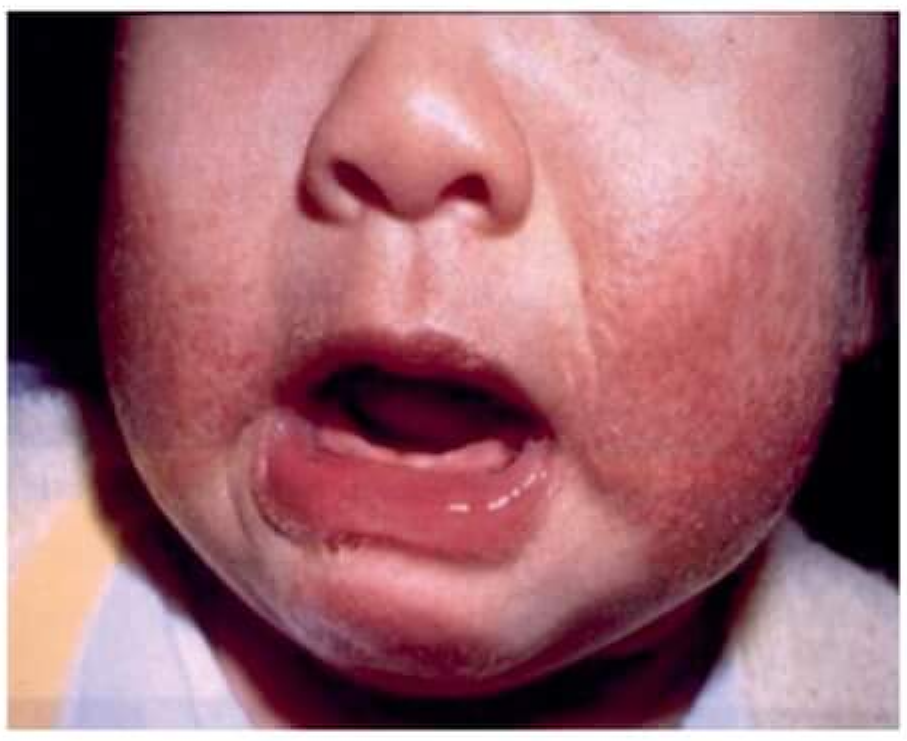
- Histology:
  - Acute- Spongiosis (oozing out lesion)
  - Chronic- Acanthosis (Lichenified lesion)
- **Treatment**

Acute	**Chronic**
-Corticosteroids (Topical + Systemic)	- Corticosteroids Topical – Calcineurin inhibitor Systemic - Azathioprine

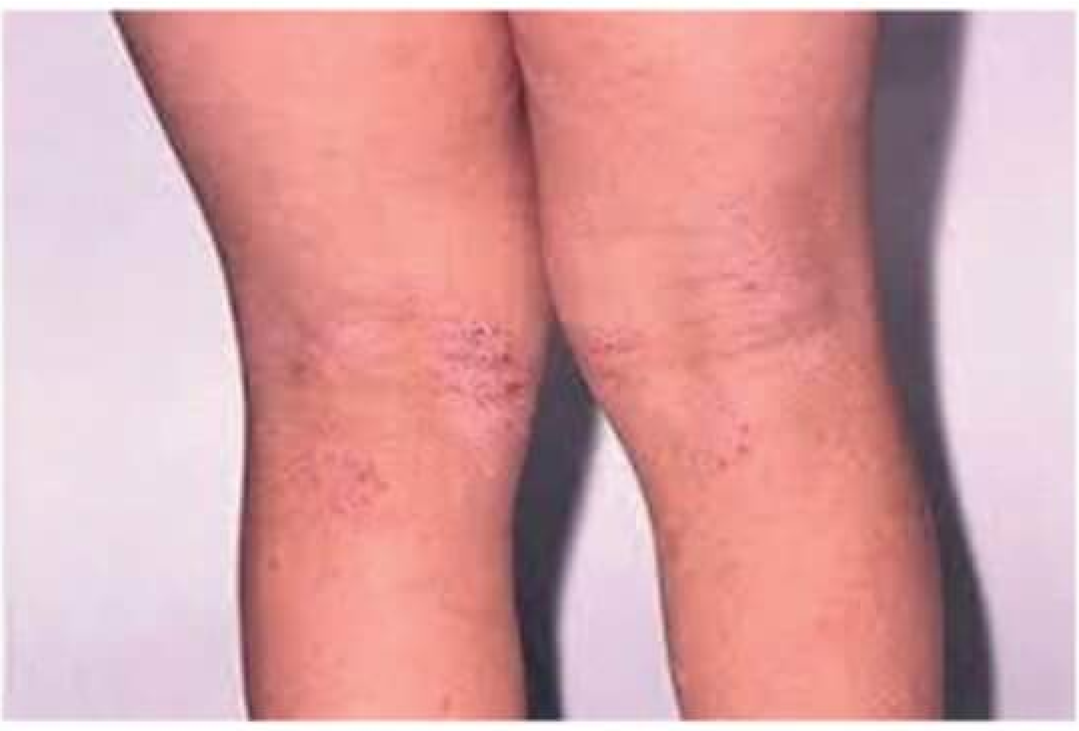
→ If Lichenified lesions- add salicylic acid
→ Secondary infection (bacterial)- staph aureus
→ Secondary infection(viral) – HSV



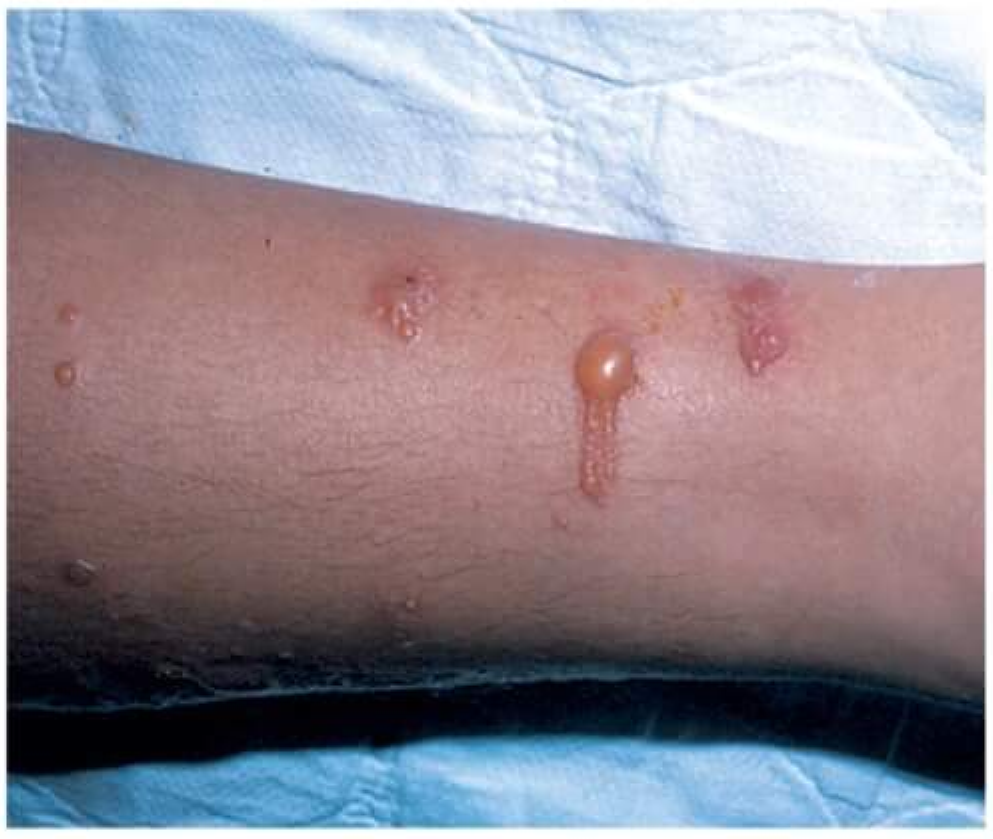
Acute eczema hand showing oozing out



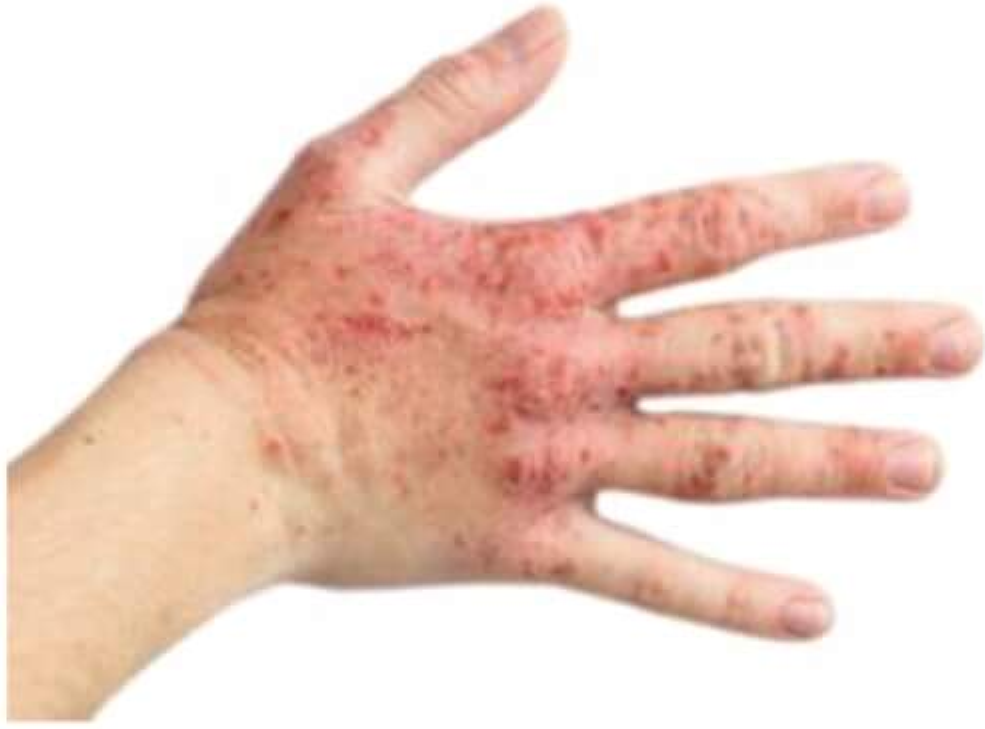
Atopic dermatitis face (cheeks)
With involvement in infantile phase
Erythematous lesions with small cracks
oozing out



Lichenified lesion of atopic dermatitis involving popliteal fossa
Hypertrophy with prominent markings.

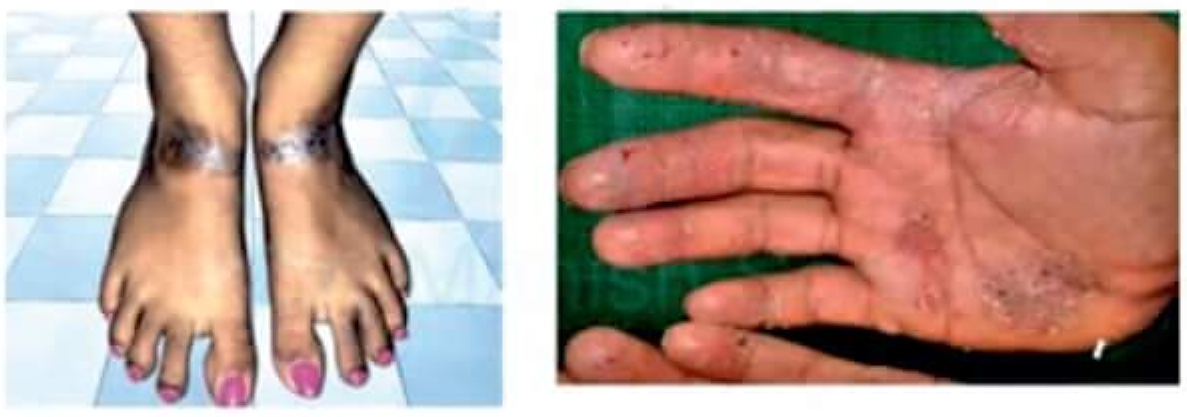


Irritant contact dermatitis with acute (exudative lesions)
And with bulla formation

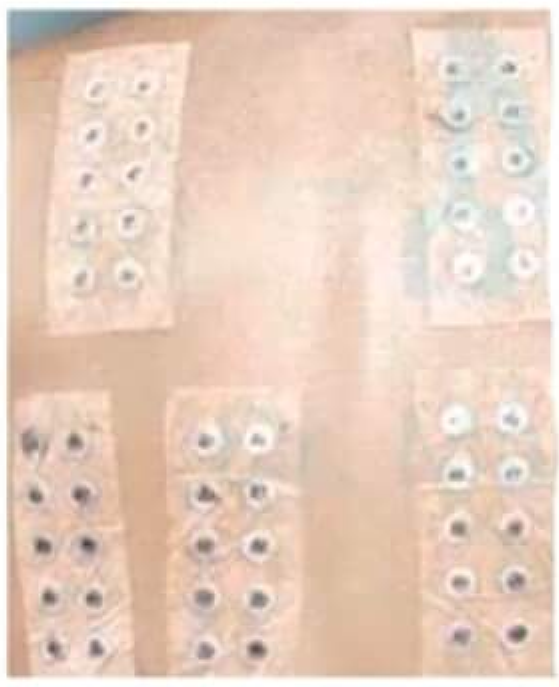


Housewives eczema with chronic (dry) lesions

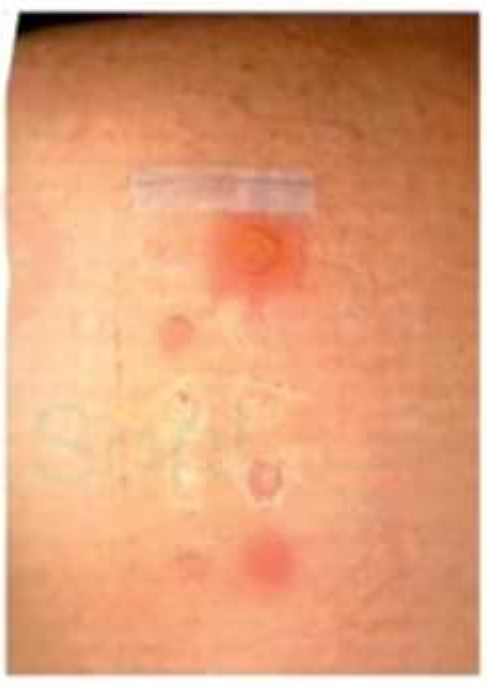
Allergic contact dermatitis due to:
A. Footwear B.cement



Allergens placed over back of the patient in finn chambers during patch testing

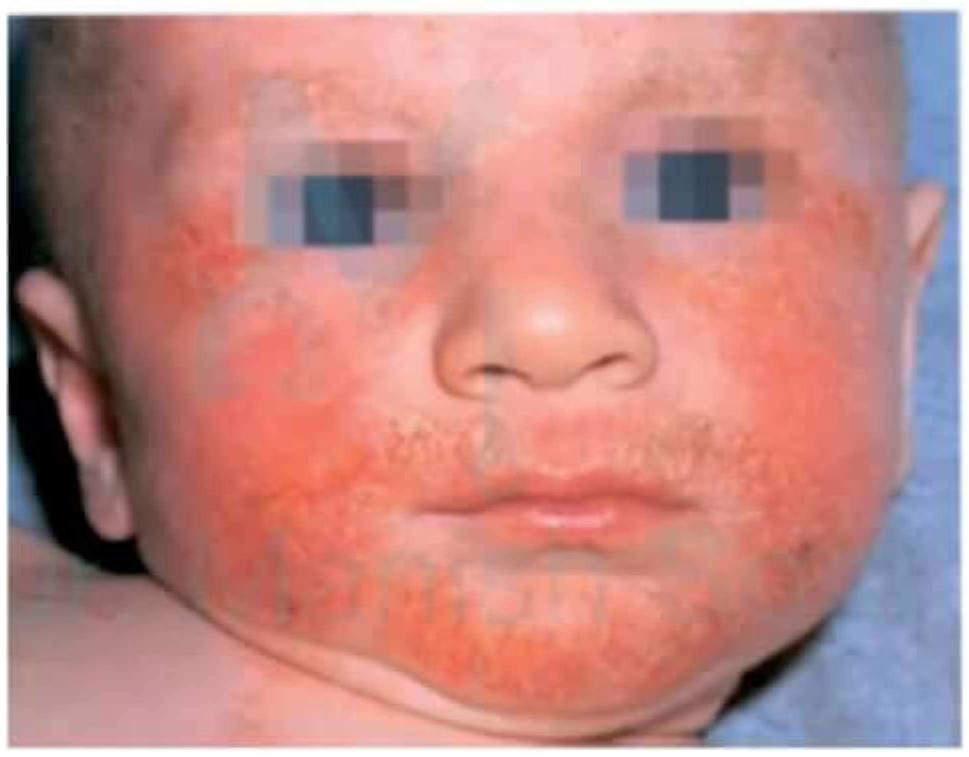


Patch testing over back of a patient with allergic contact dermatitis showing strong positive reaction

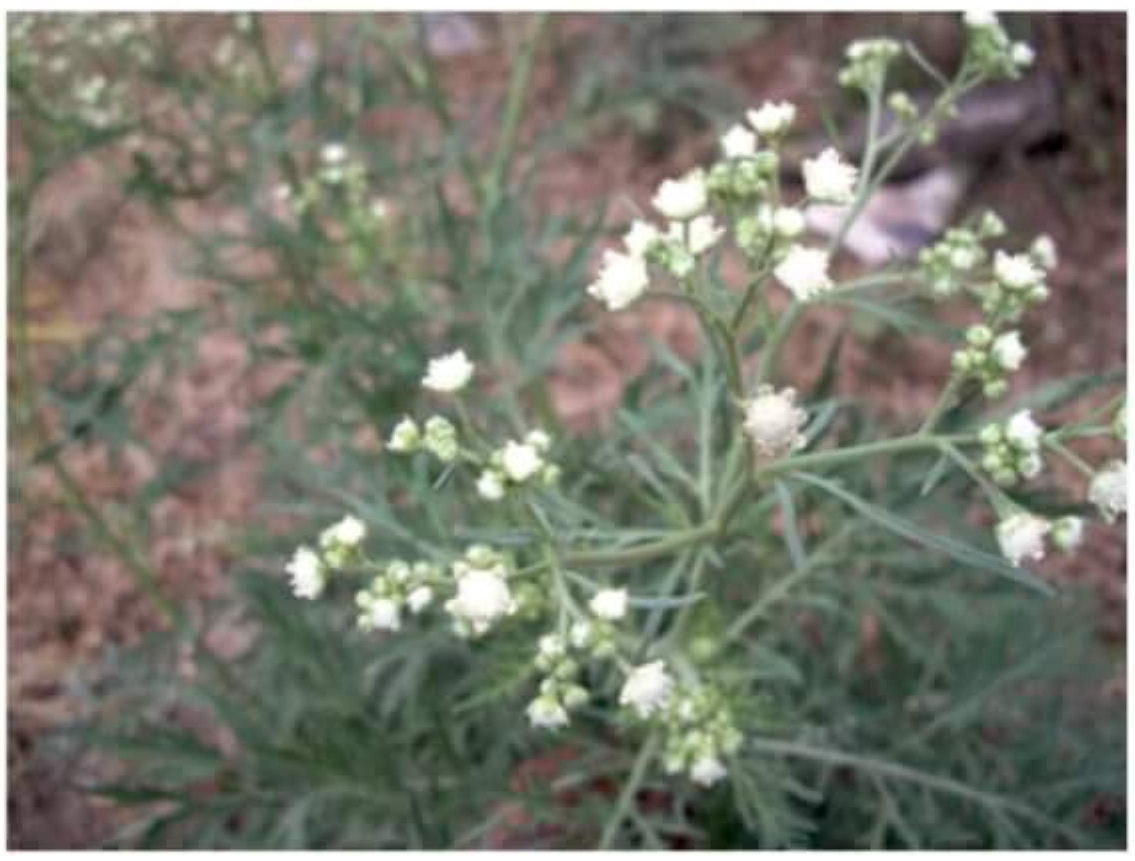


Patch test-

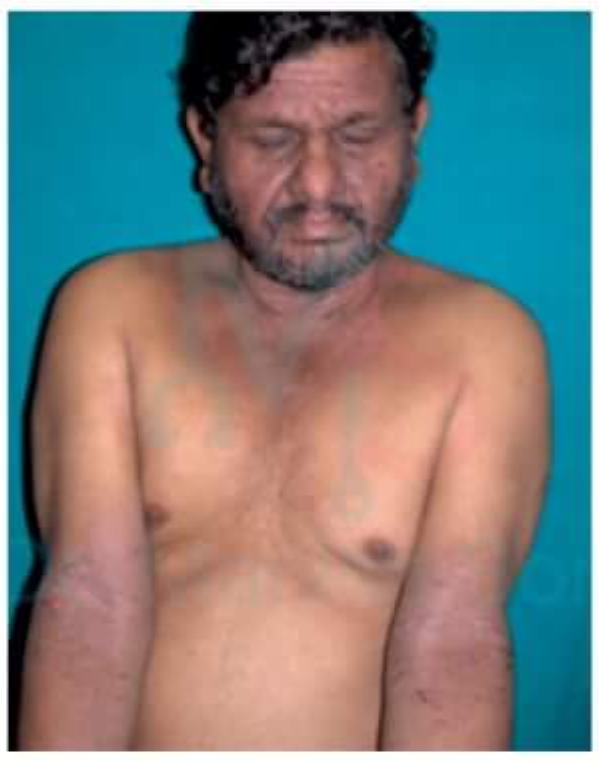
Reading done on 2nd day (48 hours) and 4th day (96 hours).



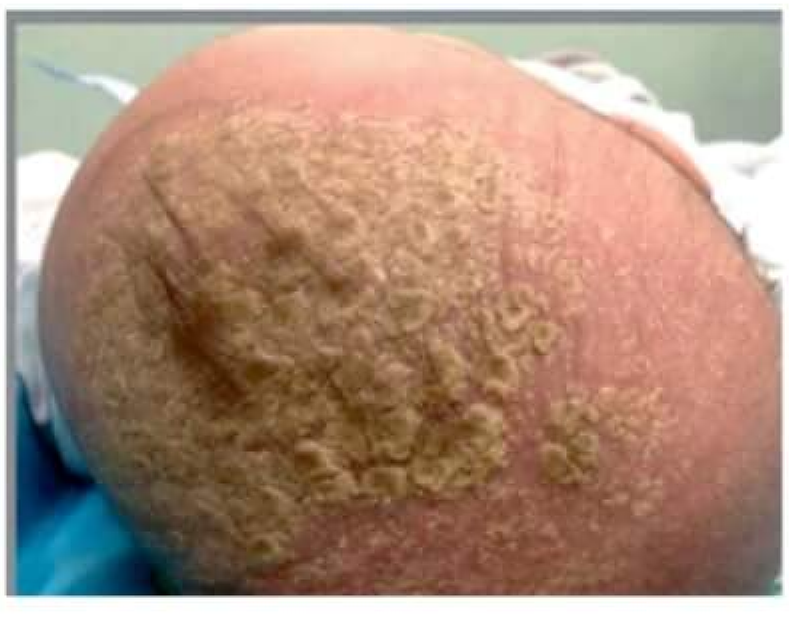
Acute eczema (atopic dermatitis) in infant's face -oozing out lesion



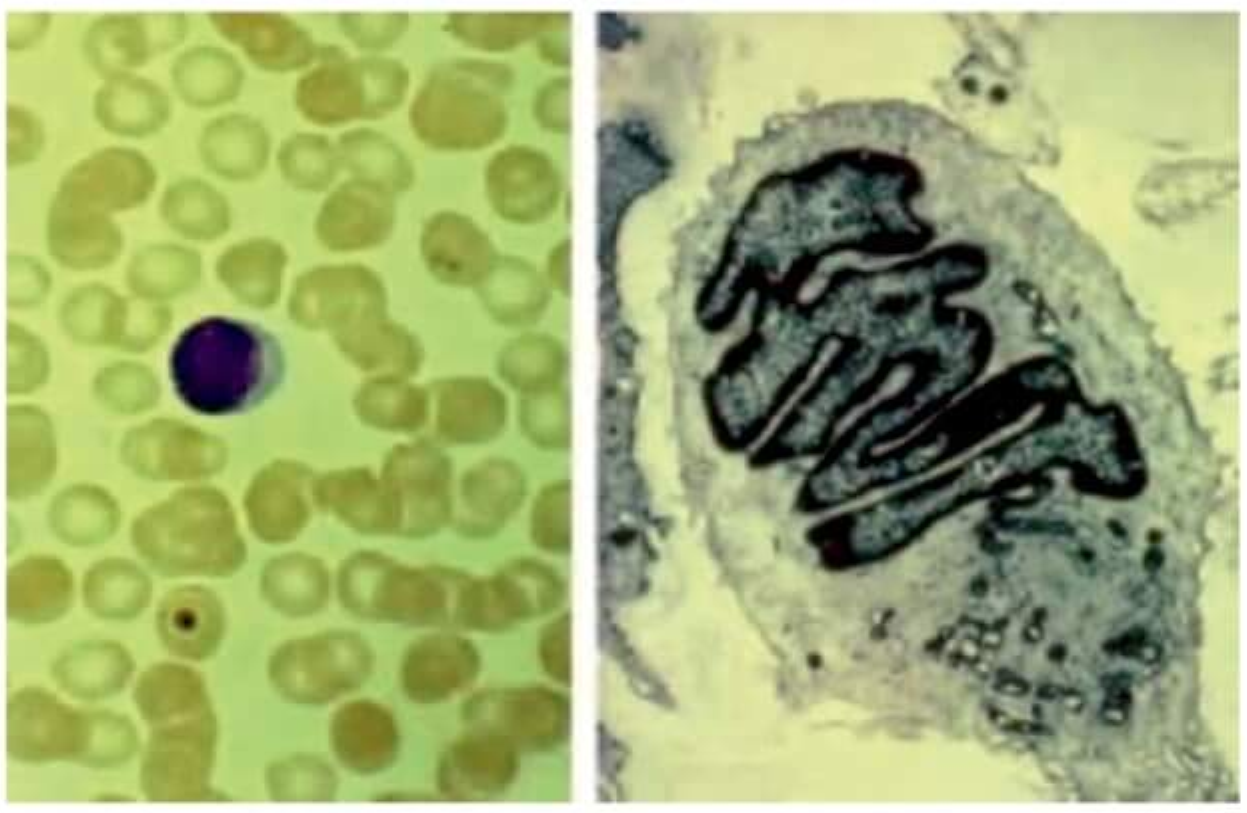
Parthenium hysterophorus (Congress grass) cause Airborne contact dermatitis In exposed areas.



- V area of neck involved.
- Exposed forearm areas
- Face
- Dorsum of hand



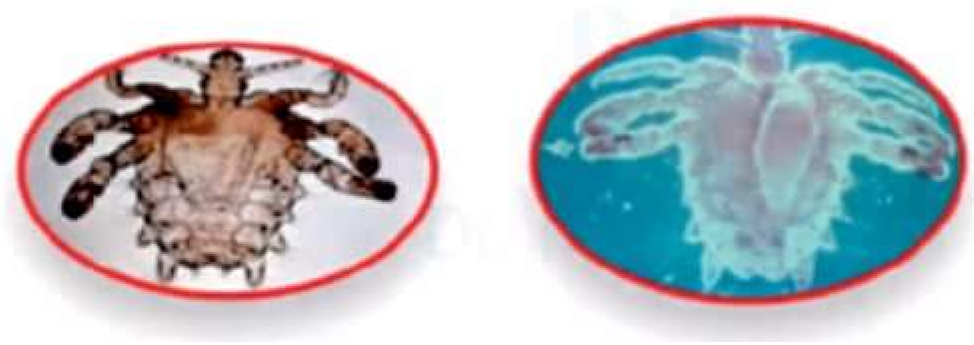
Greasy scaling seen in Cradle cap-Seborrheic dermatitis of infant



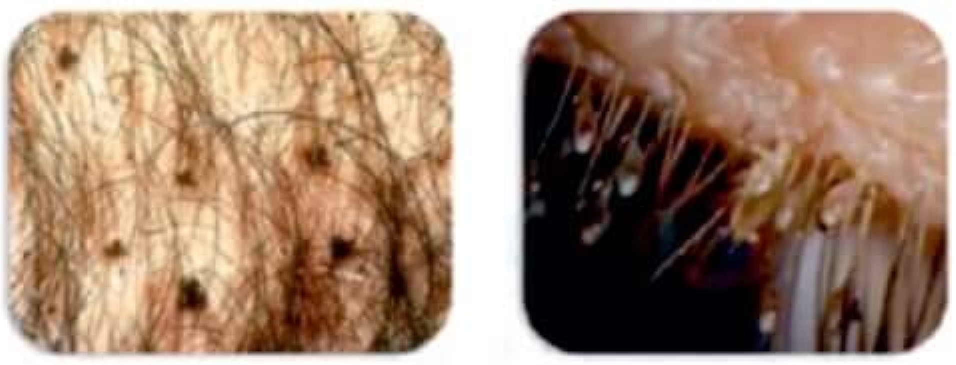
Sezary cells in peripheral blood showing features of atypical cerebriform nucleus

Infestations

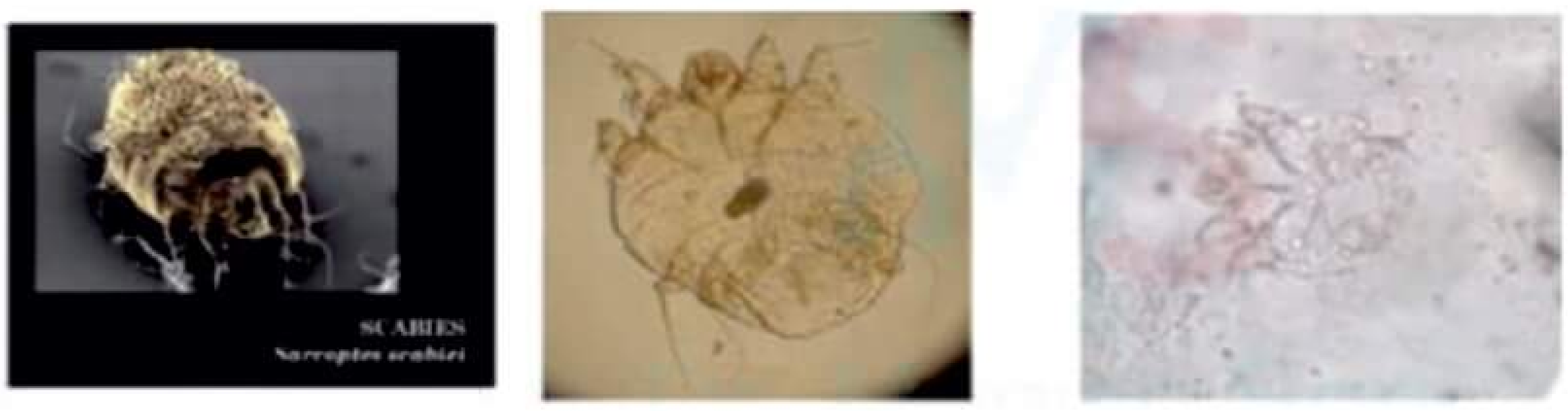
Pubic lice/ crabs



lice in pubic area

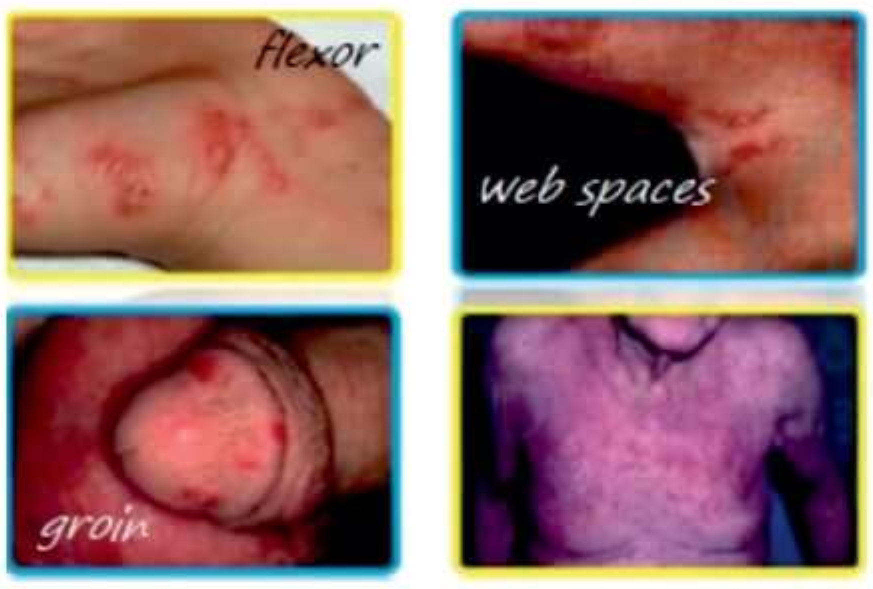


Scabies Q

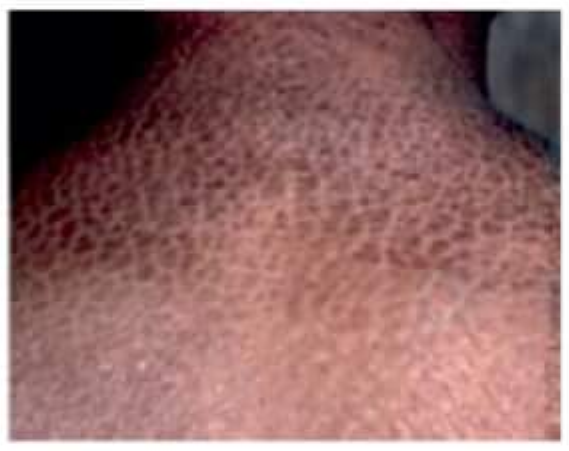


Sarcoptes scabiei var hominis: eyeless mites with four pairs of legs (two pairs in front and two pairs Behind)

Common sites involved in scabies

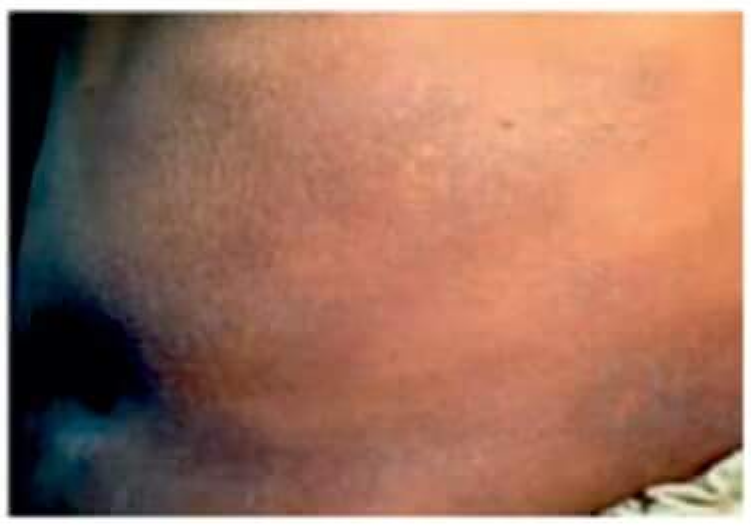


Ichthyosis

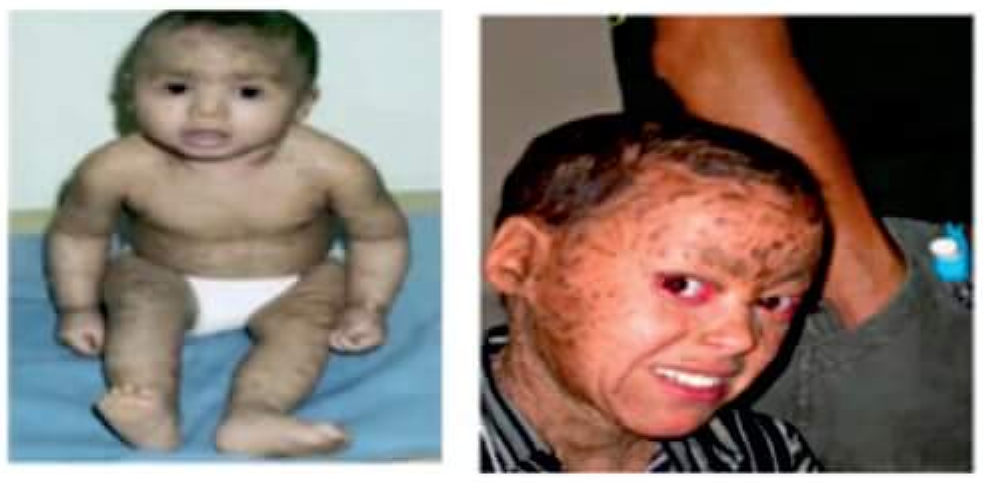


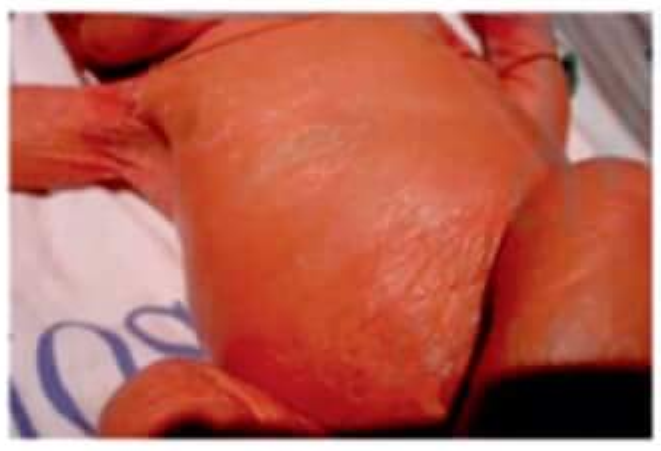
Ichthyosis vulgaris
(fish like scales and severe dryness of skin)

X-linked ichthyosis

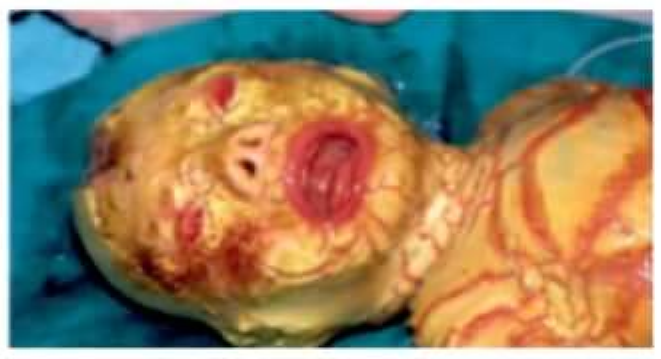


Lamellar ichthyosis





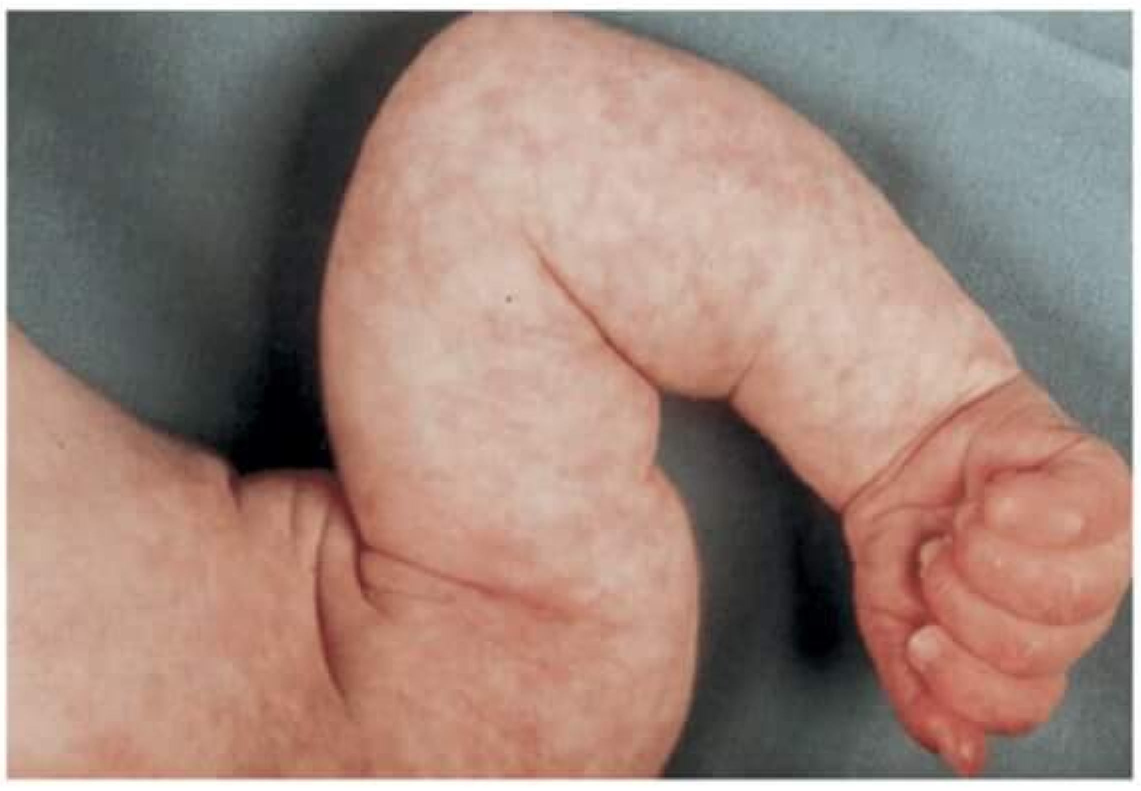
Collodion baby



Harlequin (type of ichthyosis)

Important fact:

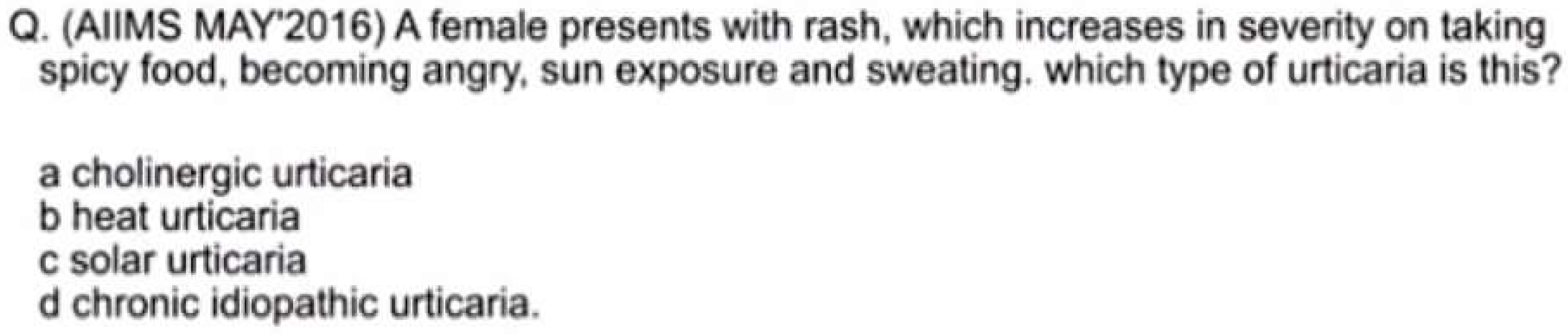
1) Cutis marmorata- bluish reticulate pigmentation seen in neonates as a physiological response to cold disappears on warming.



2) Erythema ab igne

- → Erythematous red reticulate pigmentation.
- → Due to constant exposure to heat.

Questions



Q. (AIIMS MAY'2016) A female presents with rash, which increases in severity on taking spicy food, becoming angry, sun exposure and sweating. which type of urticaria is this?

a cholinergic urticaria
b heat urticaria
c solar urticaria
d chronic idiopathic urticaria.

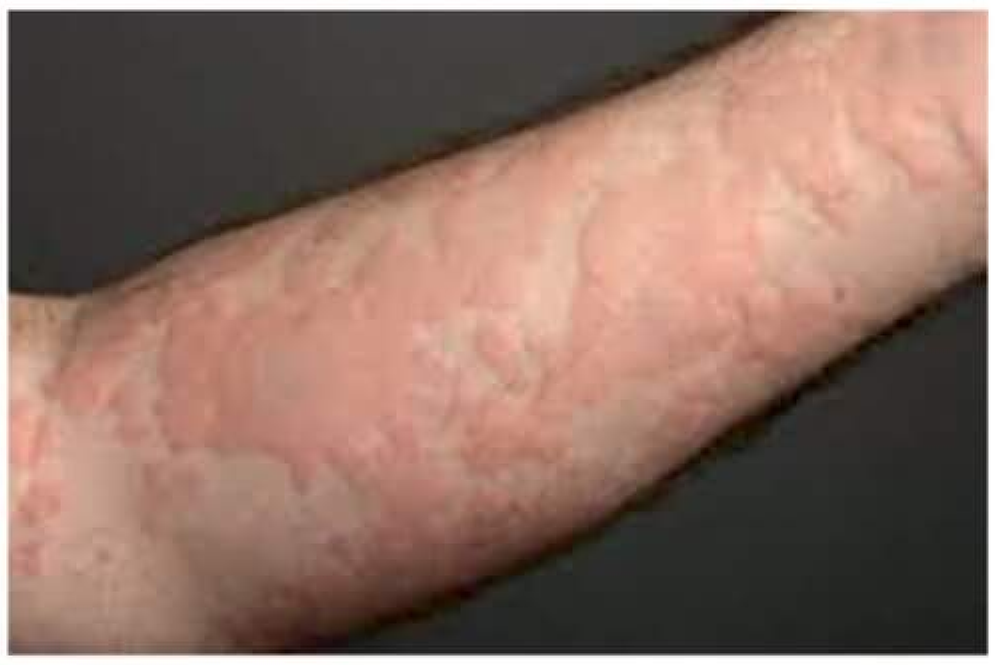
Answer- cholinergic urticaria

3.Urticaria:

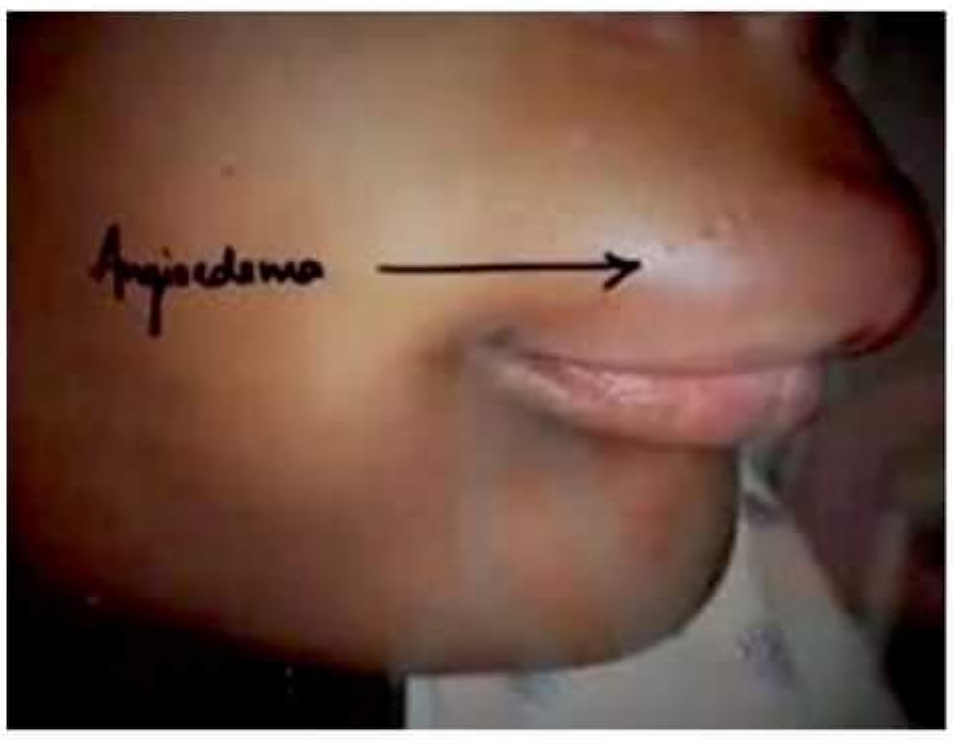
Acute
(6 weeks)

Chronic
1) Spontaneous:
   i) idiopathic
   ii)autoimmune
2)Inducible:
   Heat, sun, sweating

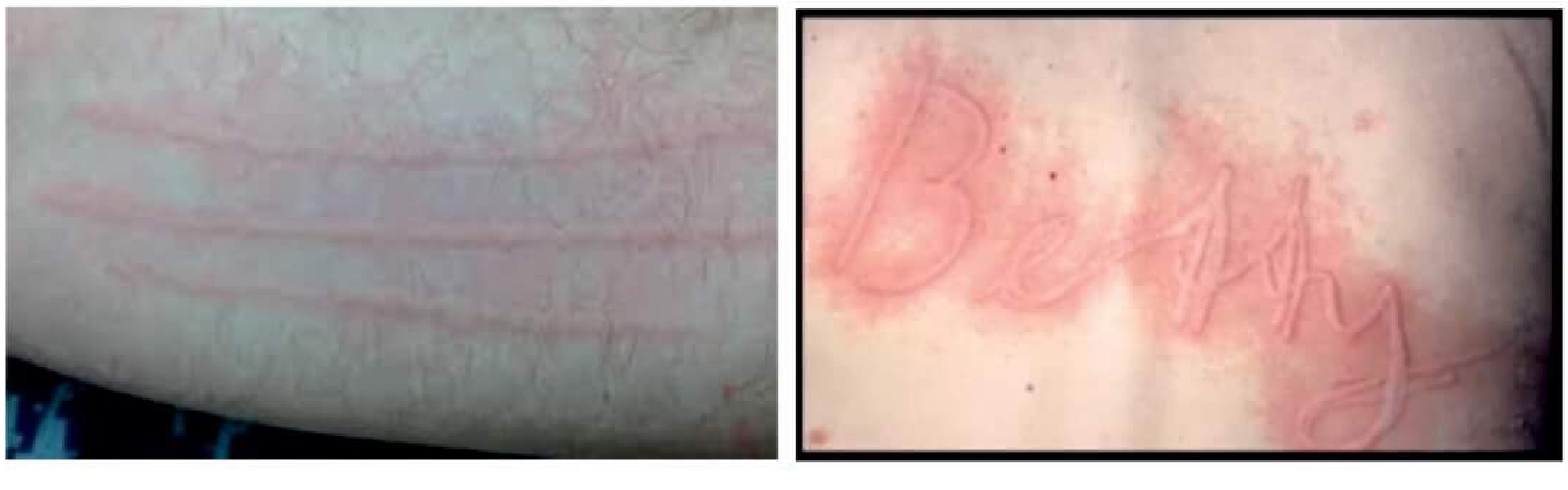
wheals (lasts for >24hrs, itchy)

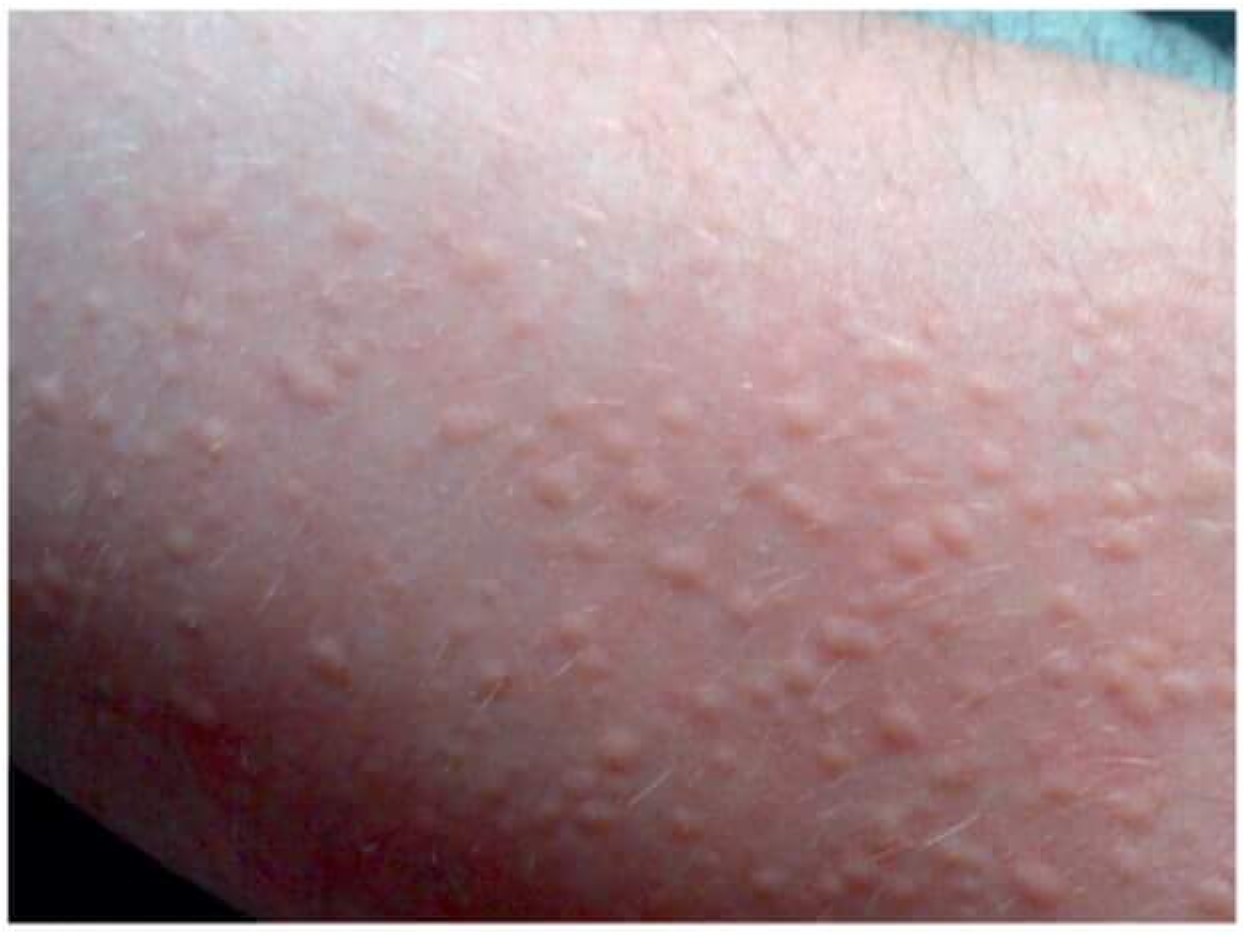


Angioedema (72 hrs painful) – subcutaneous dermal swelling.



Dermographism in urticaria – elevation of skin due to scratch





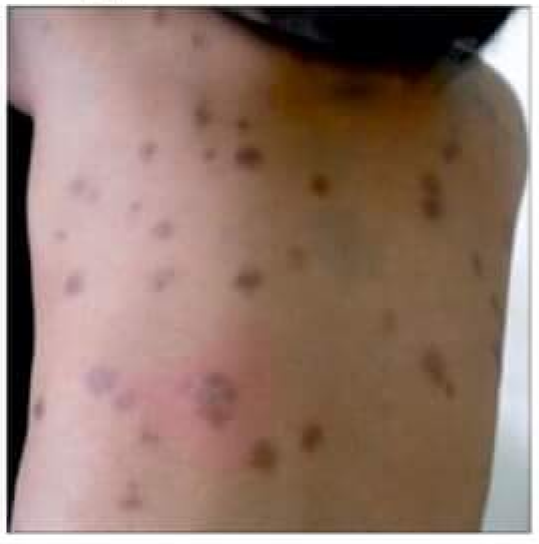
Cholinergic urticaria

- Wheals – transient, <24hrs (if >24 hrs urticarial vasculitis), dermal edema
- Angioedema – (quincke edema) swelling, non itchy, painful, deeper
- Hereditary angioedema- sudden , familial, no urticaria.
  c1 esterase inhibitor protein inhibit kinin.

  Triggered by – activation of factor 12 (hageman) –consumes inhibitor-kinin level increase – angioedema .

  Low complement levels (c4,c1,c2)
- Cholinergic urticaria is one of the physical urticarias brought on by a physical stimulus. Although this stimulus might be considered to be heat, the actual precipitating cause is sweating.
- ETIOLOGY
  Mast cells seem to be critically involved in cholinergic urticaria. Serum histamine, the principal mediator, rises in concentration with experimentally induced exercise, accompanied by eosinophil and neutrophil chemotactic factors and tryptase. A reduction of the alpha1-antichymotrypsin level, is present.
- Possible allergy-based etiology
  * increased incidence in patients with atopic dermatitis (AD)
  *positive immediate sensitivity to sweat.
  *Most patients demonstrate immediate-type skin responses to their own sweat and satellite wheals after acetylcholine injection.

Urticaria pigmentosa -mutation in c-kit gene
→ type of cutaneous mastocytosis
→ Darier's sign- also seen in histiocytosis, leukemia, xanthogranuloma



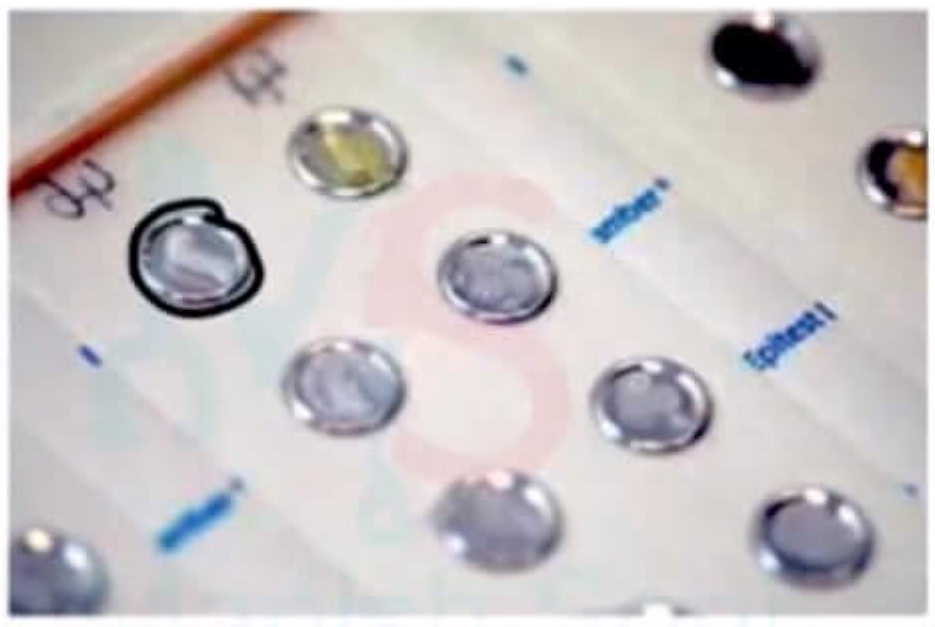
Cutaneous Mastocytosis
-Dariers sign +

Q. (AIIMS MAY'2016) A farmer presents with lesions on face , V of neck, arms. which increased on severity in summer and decrease in winters. which test will confirm the diagnosis?

a skin biopsy
b patch test
c igA levels in blood
d prick test

ANS patch test

Answer- Patch test for airborne contact dermatitis



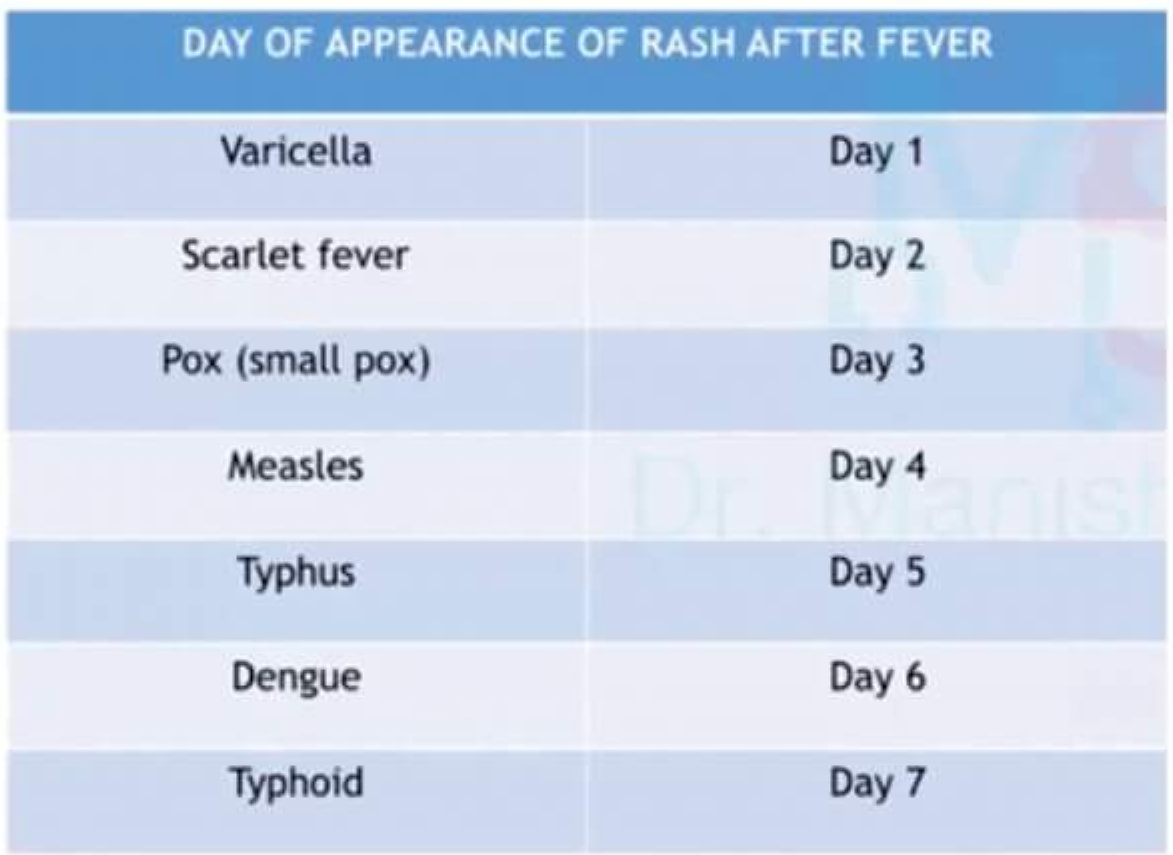
Dr. Manish Soni

- Patch tests is done to find out whether their skin condition may be caused or aggravated by a contact dermatitis.
- Reading: 48 and 96 hour.

  Negative (-)
  Irritant reaction (IR)
  Equivocal / uncertain (+/-)
  Weak positive (+)
  Strong positive (++)
  Extreme reaction (+++)

- Prick tests, are used to diagnose hay fever allergy (house dust mit grass pollens and cat dander). Skin prick tests have very limited value.

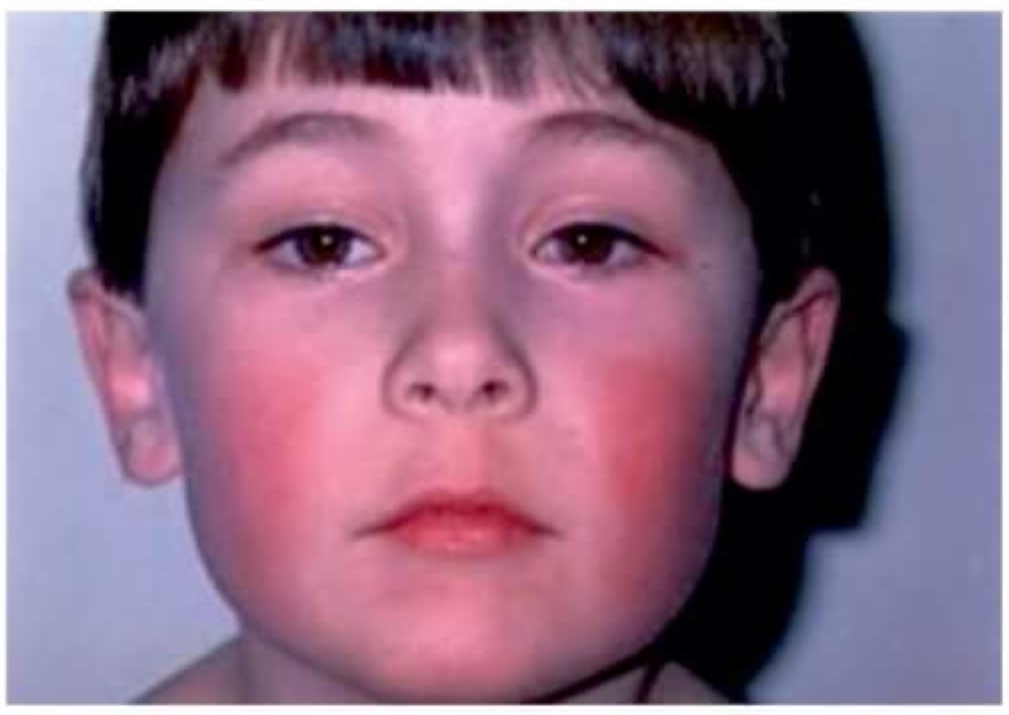
Mnemonic: Very sick person must take double treatment

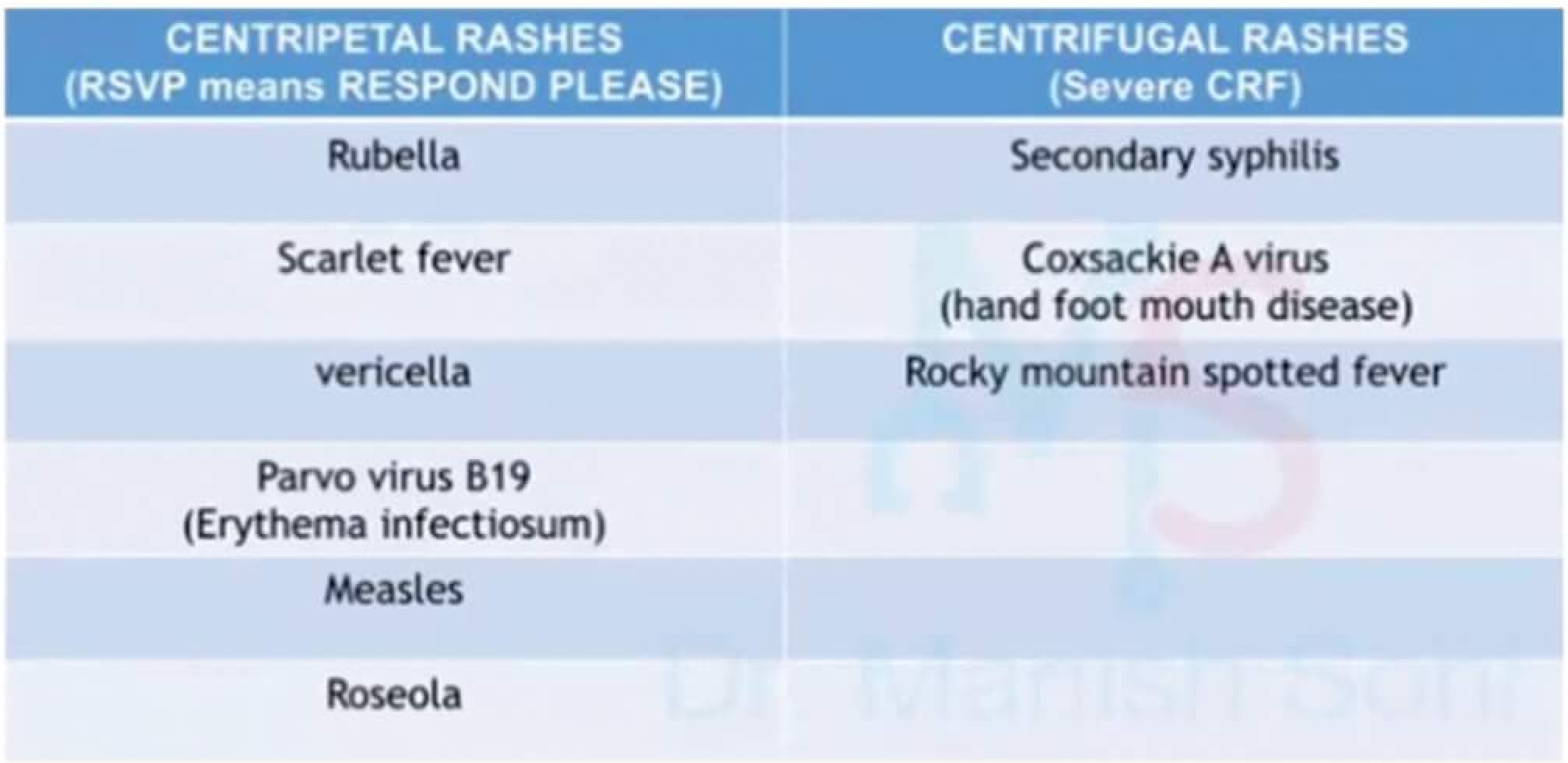


DAY OF APPEARANCE OF RASH AFTER FEVER	
Varicella	Day 1
Scarlet fever	Day 2
Pox (small pox)	Day 3
Measles	Day 4
Typhus	Day 5
Dengue	Day 6
Typhoid	Day 7

Erythema infectiosum

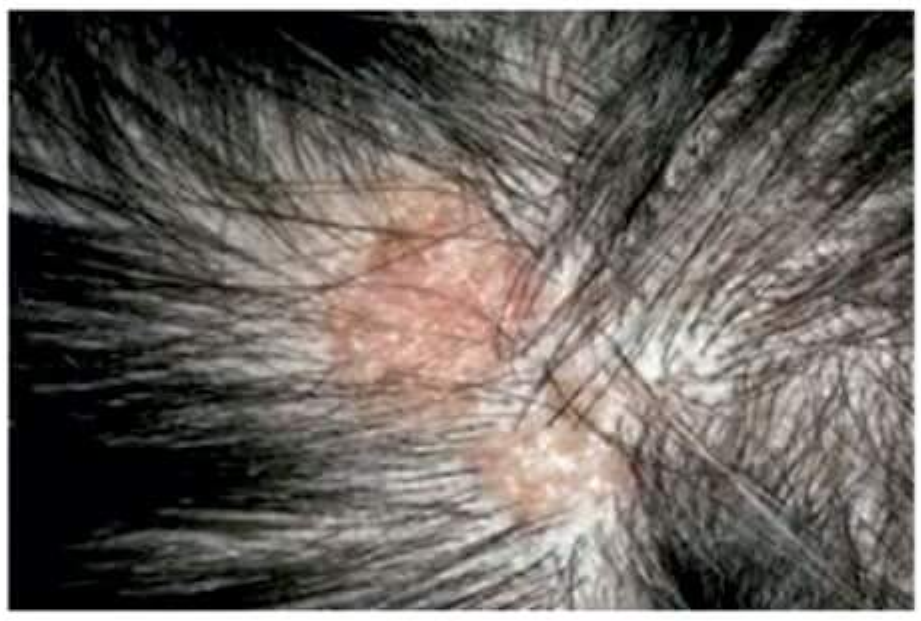
-erythematous lesion present over cheek called as slapped cheek appearance





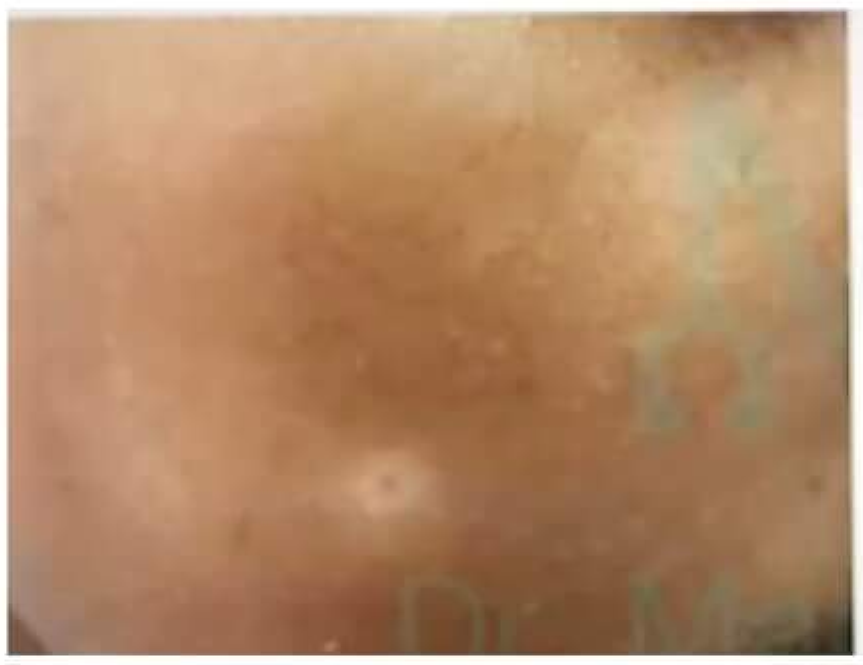
CENTRIPETAL RASHES (RSVP means RESPOND PLEASE)	CENTRIFUGAL RASHES (Severe CRF)
Rubella	Secondary syphilis
Scarlet fever	Coxsackie A virus (hand foot mouth disease)
vericella	Rocky mountain spotted fever
Parvo virus B19 (Erythema infectiosum)	
Measles	
Roseola	

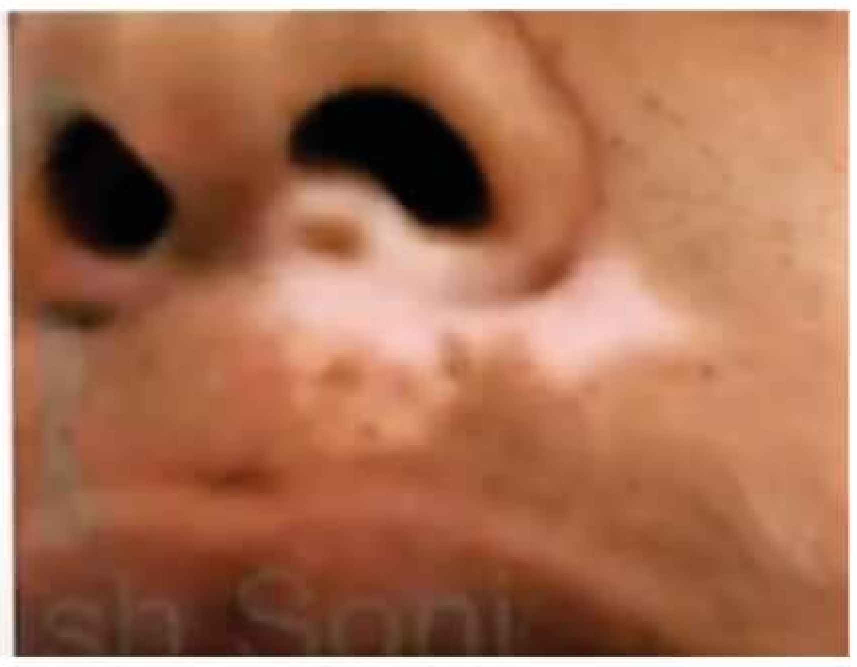
Nevus sebaceous- It has a predilection for the scalp (vertex). Solitary, hairless patch is noted on the scalp at birth or in early childhood.



Skin lesions

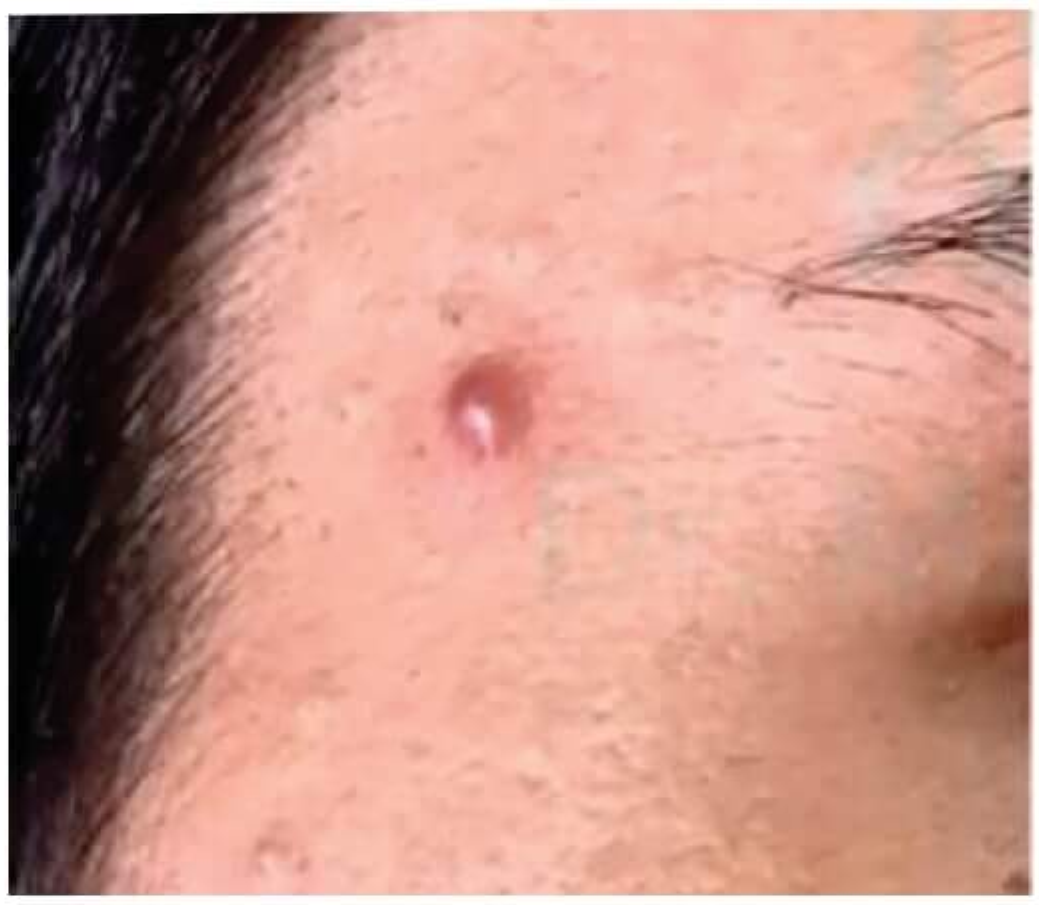
Primary lesions of skin



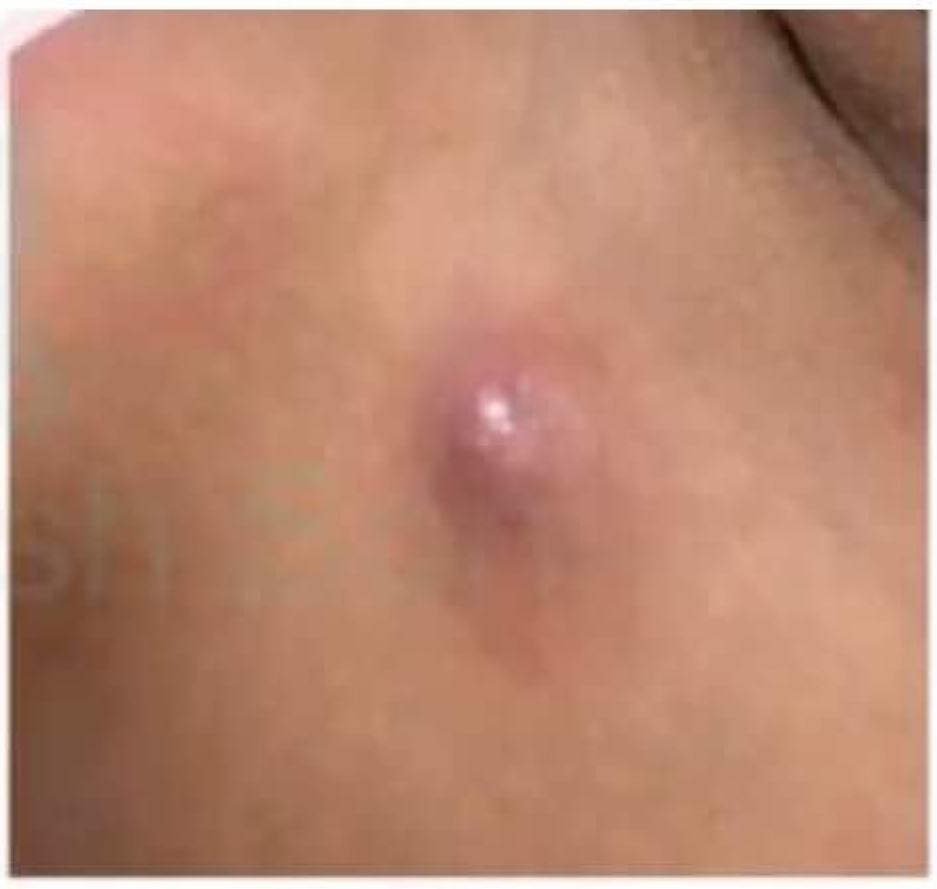


Macule		Patch
< 0.5cm		> 0.5cm
Flat	(Neither raised nor depressed)	Flat

Raised, solid lesion

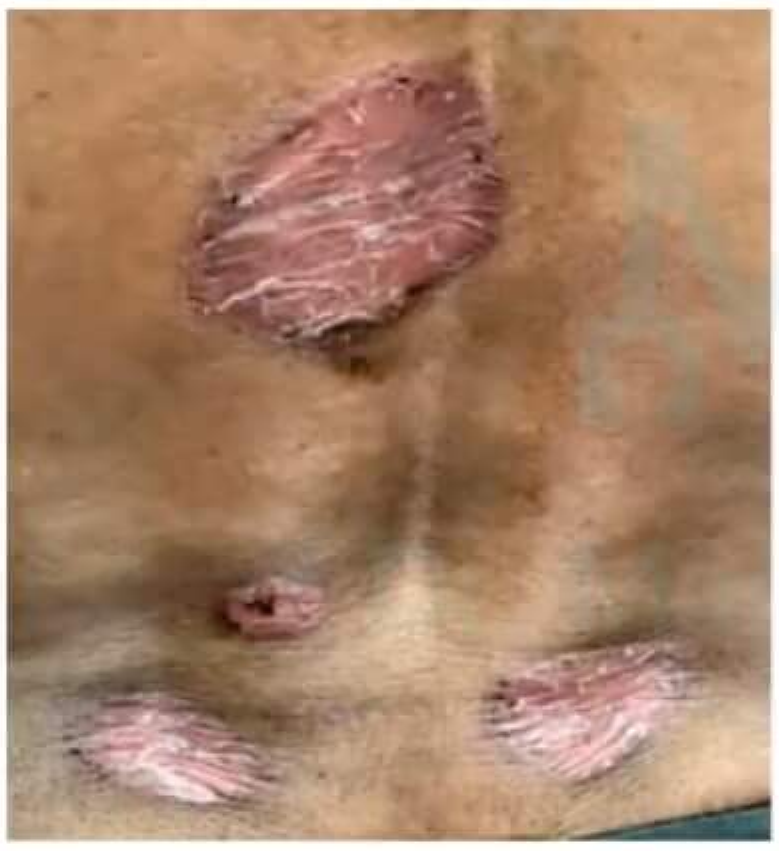


<0.5cm – papule

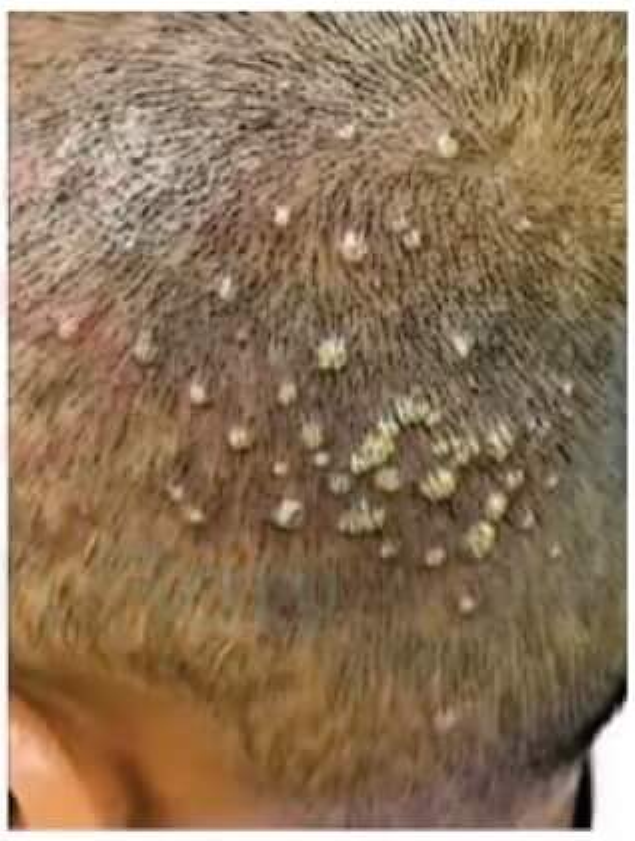


>0.5cm – nodule

Raised, solid but horizontally more

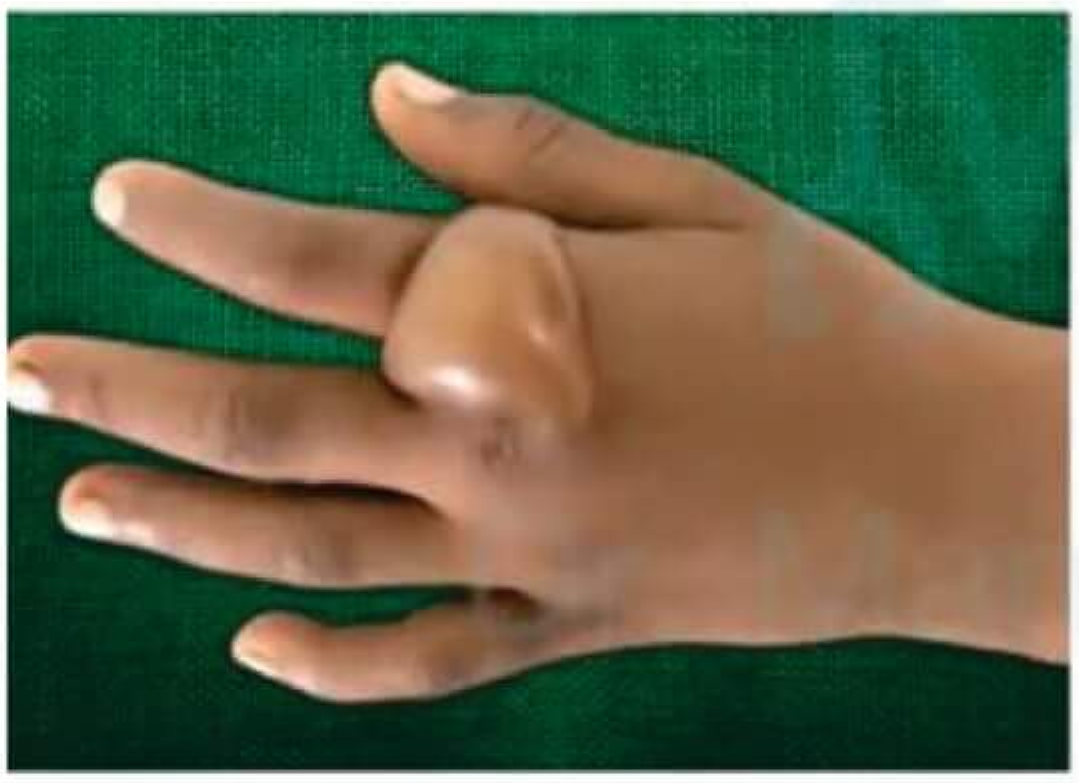


>0.5cm-Plaque

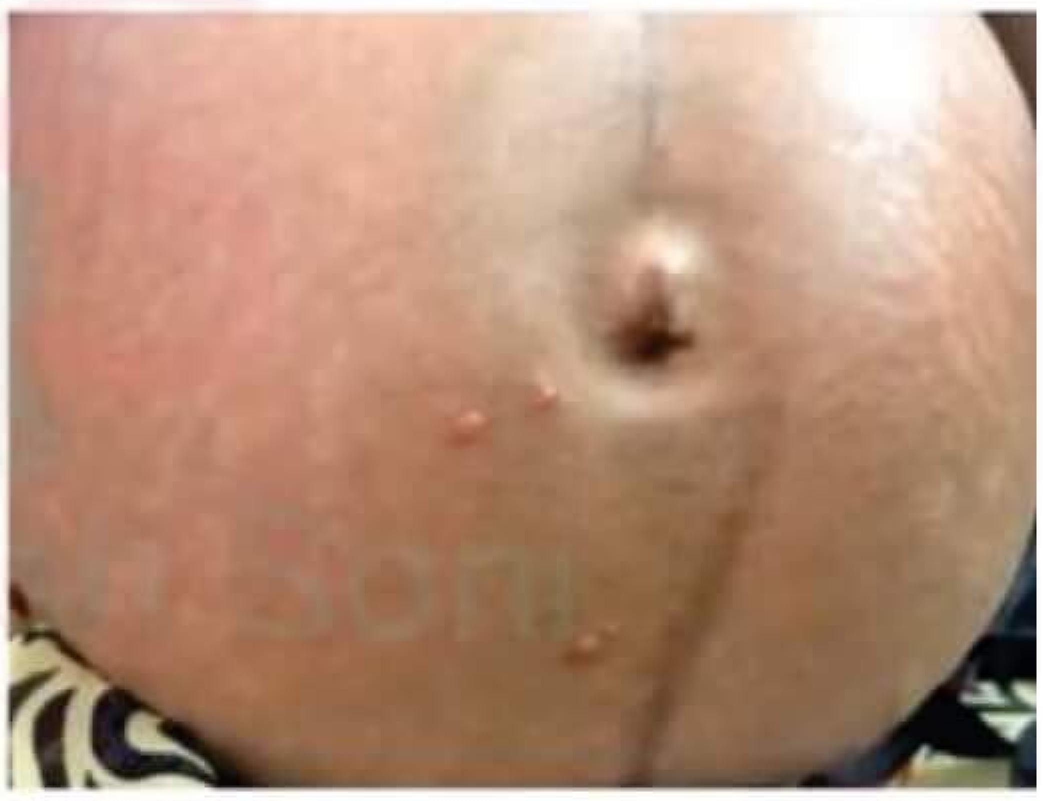


pus filled lesion-pustule

Fluid filled

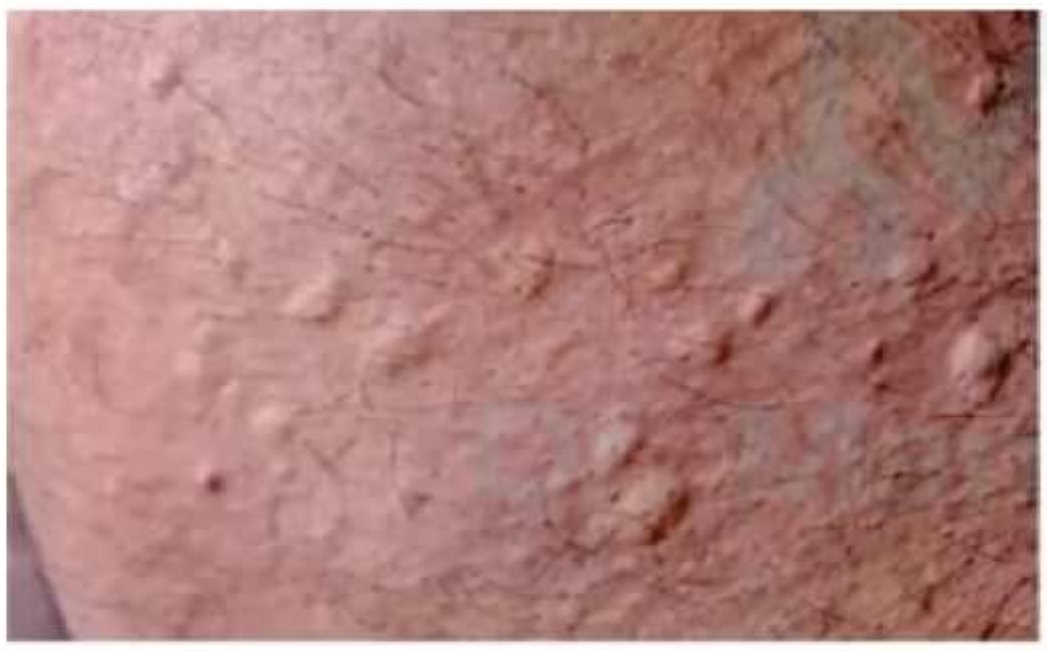


>0.5cm - bulla



<0.5cm- vesicle

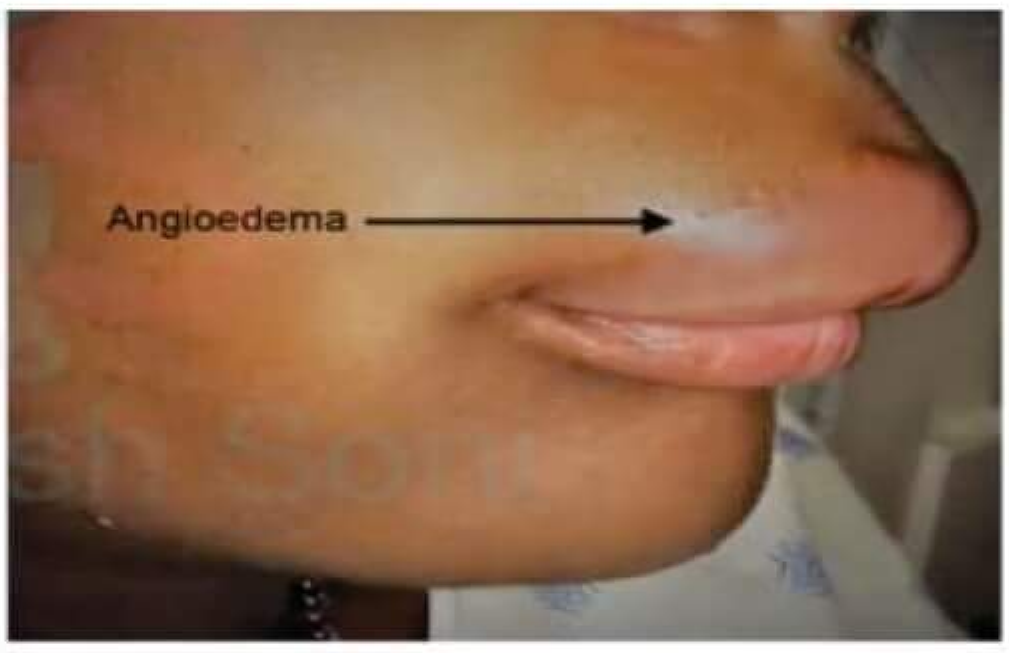
Transient and due to edema



<24 hours

Wheals or hives

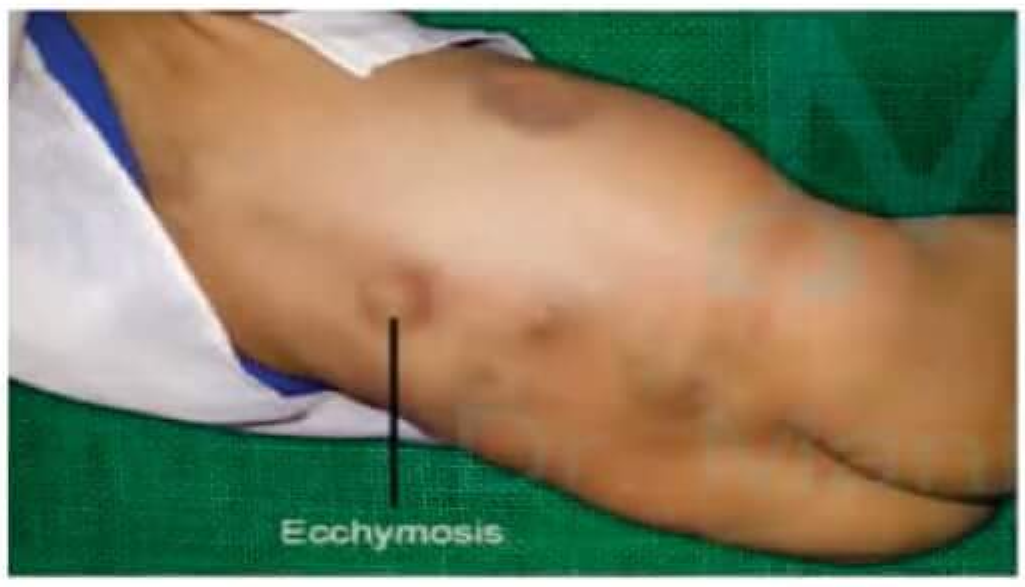
Urticaria due to accumulation

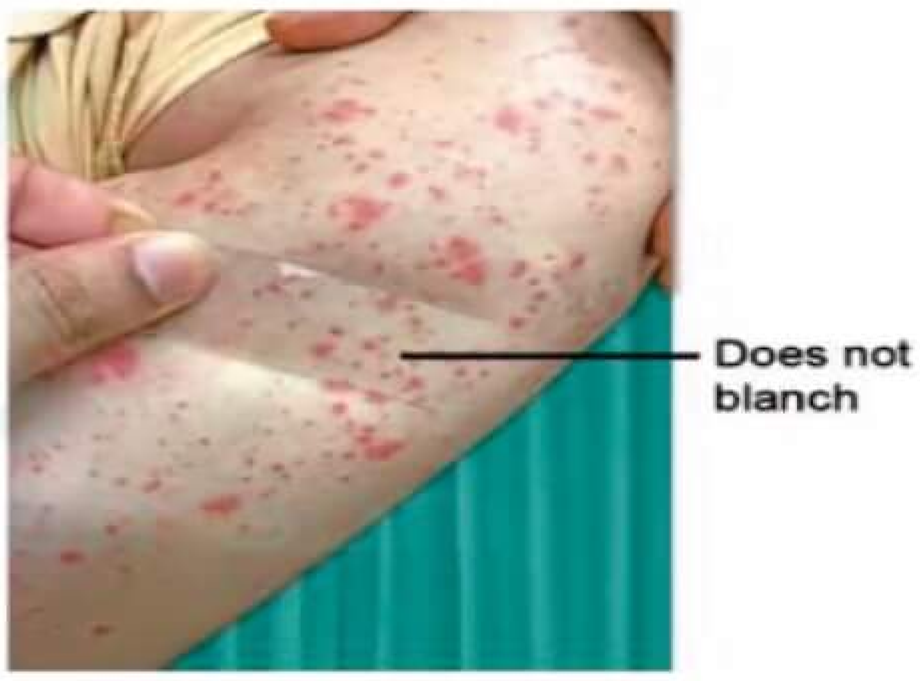


>24 hours

Due to persistant fluid accumulation in deeper layer of fluid in hypodermis.

Extravasation of blood

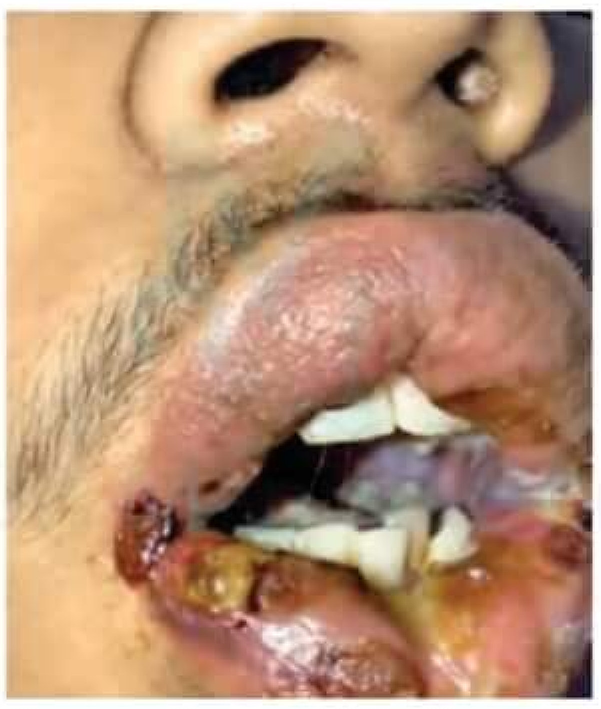




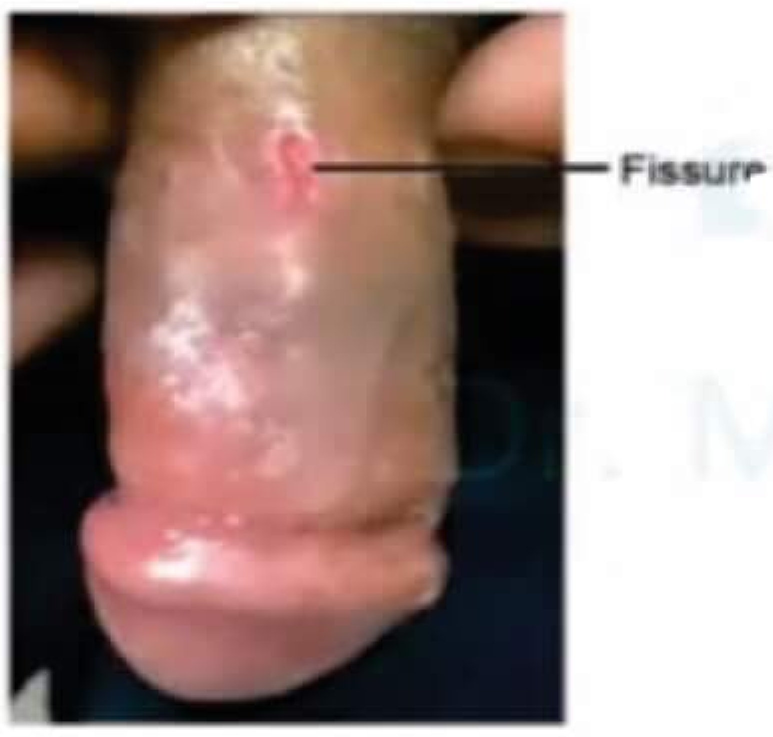
Ecchymosis(>10mm) purpura (3-9mm) Petechiae(1-2 mm)

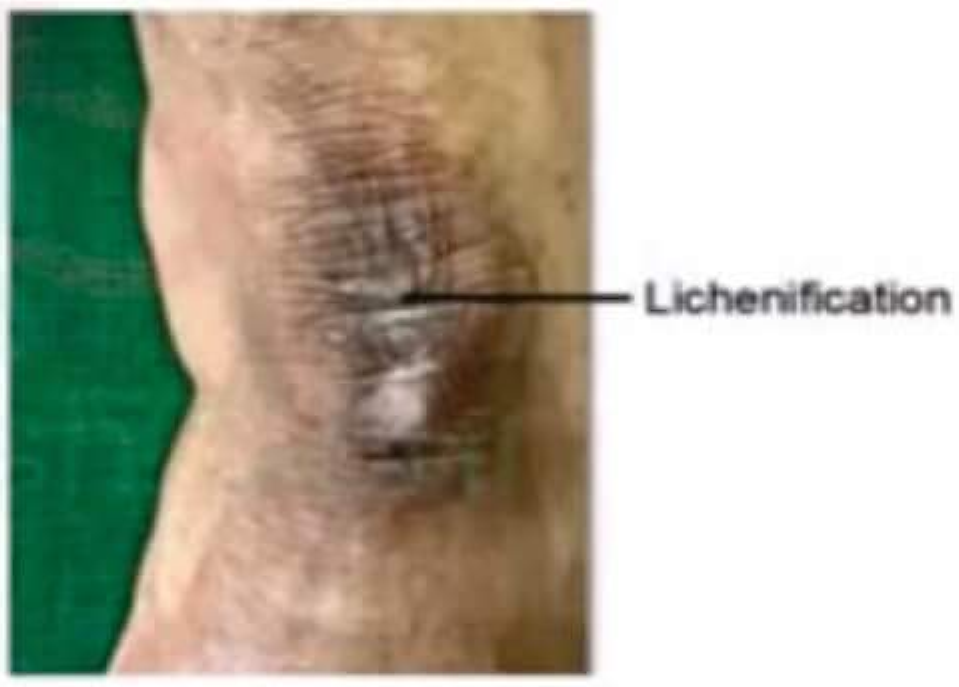
Do not blanch on diascopy

Secondary lesions



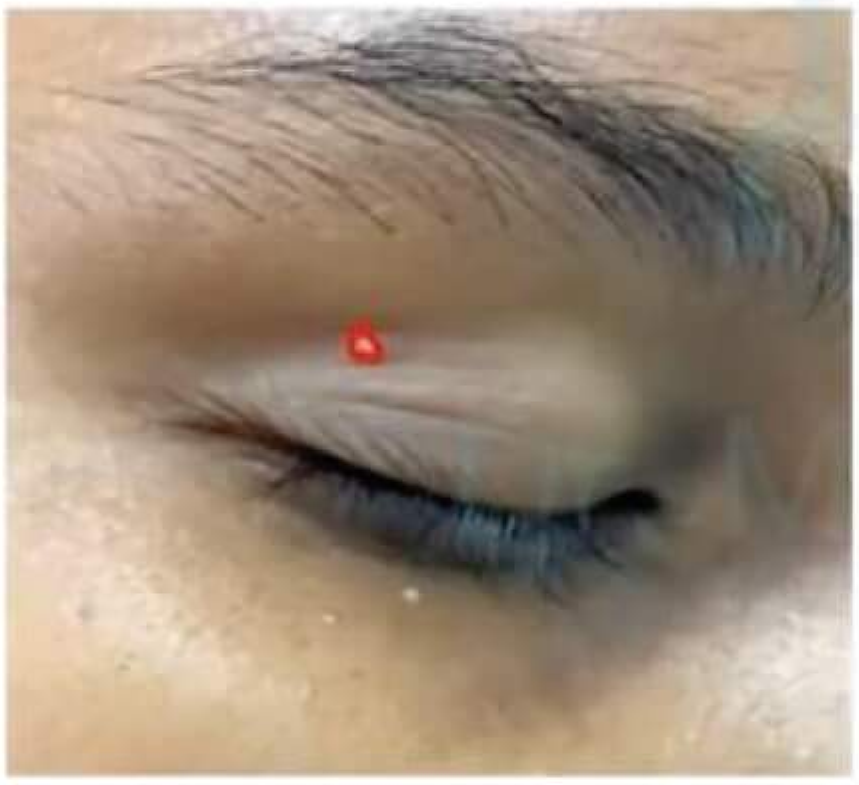
-develop over primary lesions
- crusting over mucosa + suggests SJS/TEN



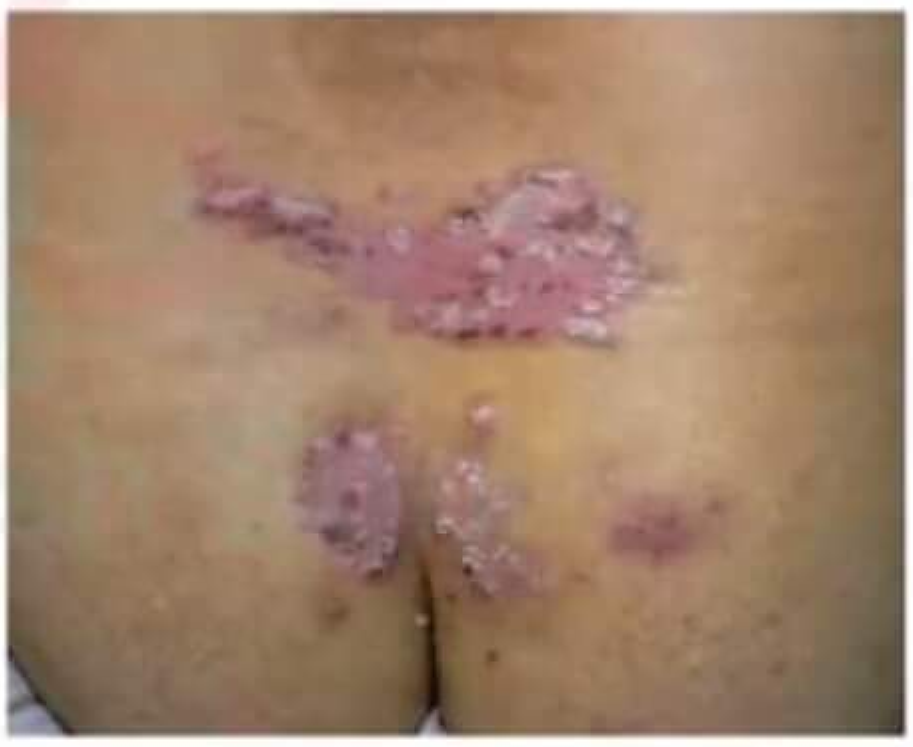


1) Skin hypertrophy
2) Increased surface markings
3) Pigmentary changes

Lichenified lesions are seen in chronic eczema

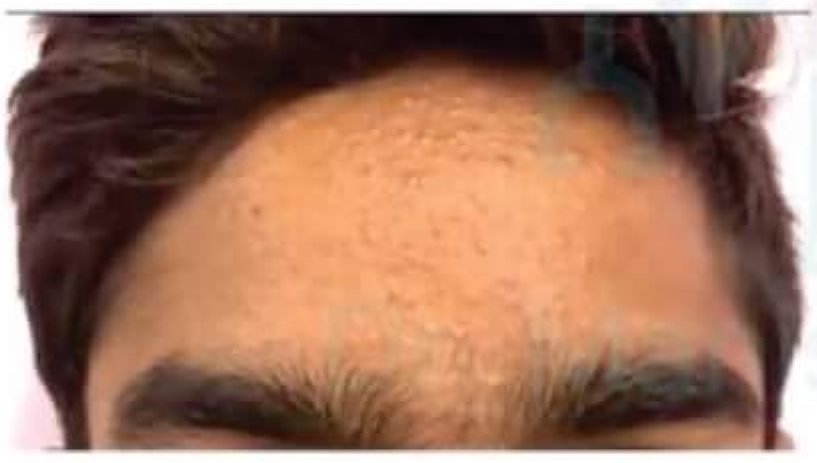


Milia -keratin cyst

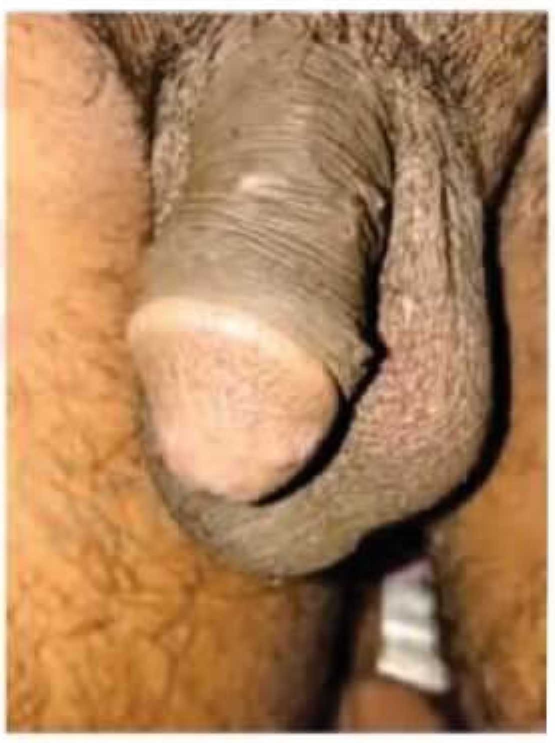


Psoriasis- erythematous plaque with scaling

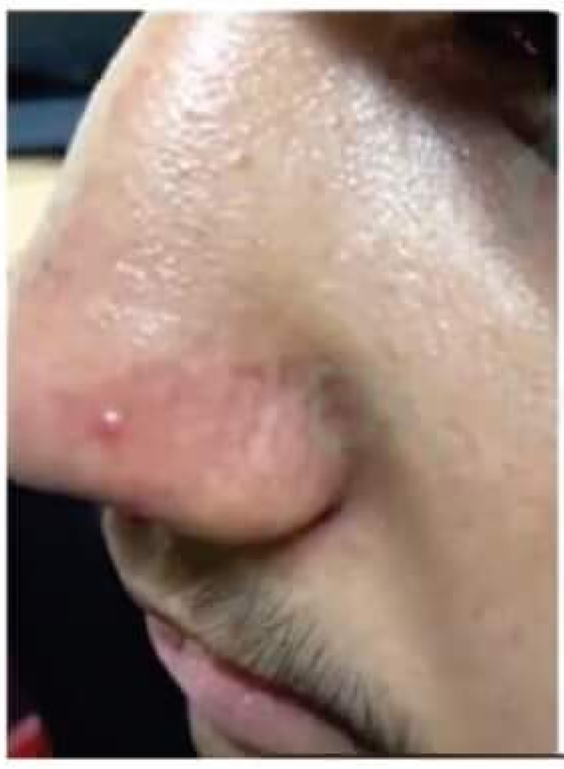
Special lesions



Acne vulgaris
-comedones

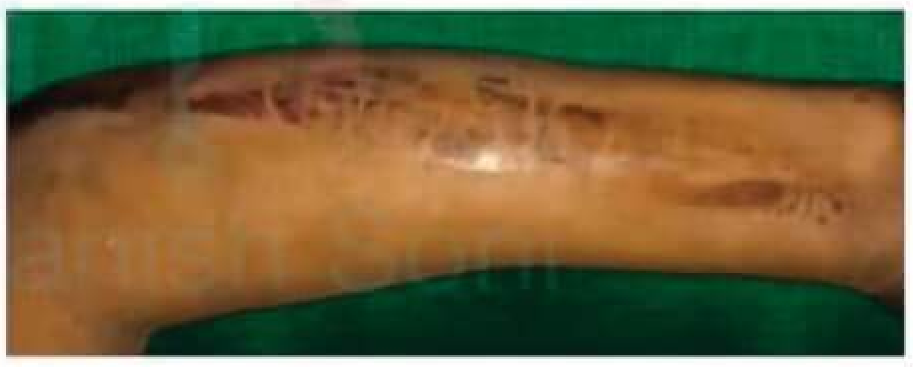


Scabies - Burrows



Poikiloderma (ATP)

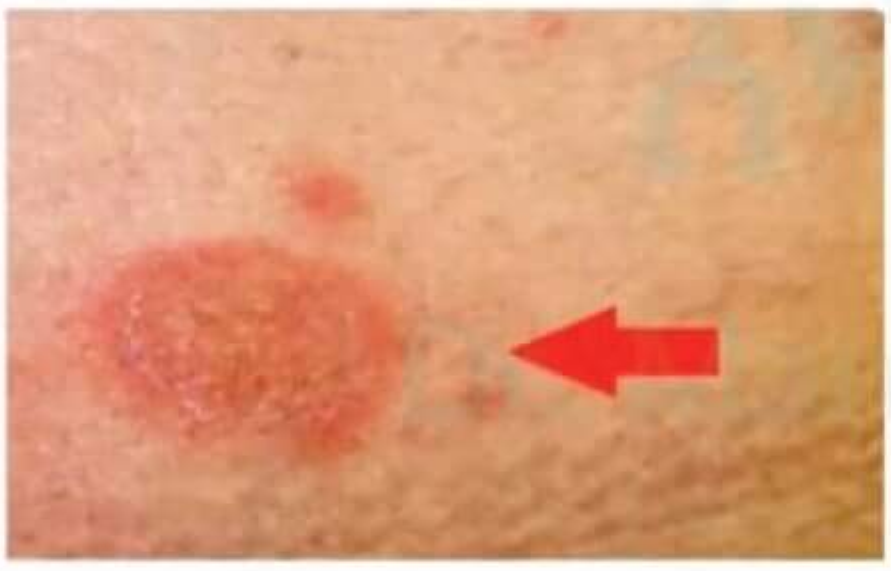
- Skin Atrophy
- Telangiectasia
- Pigmentary changes



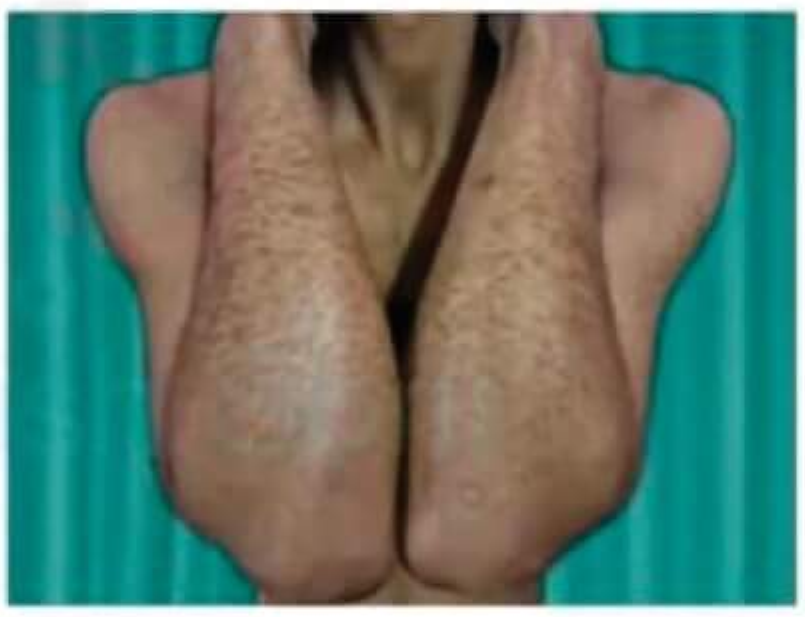
Scleroderma

-atrophy or sclerotic lesions

Collarette scales in pityriasis rosea

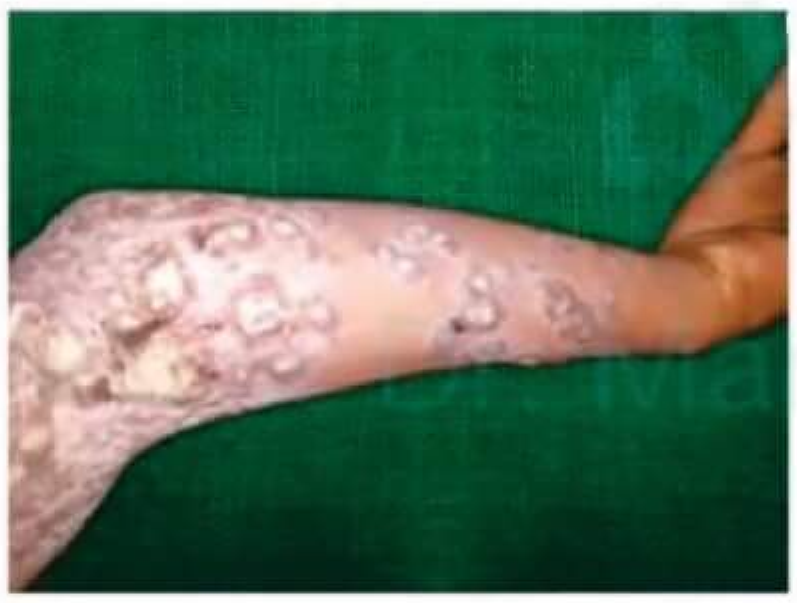


Ichthyosiform (fish like) scales of ichthyosis vulgaris

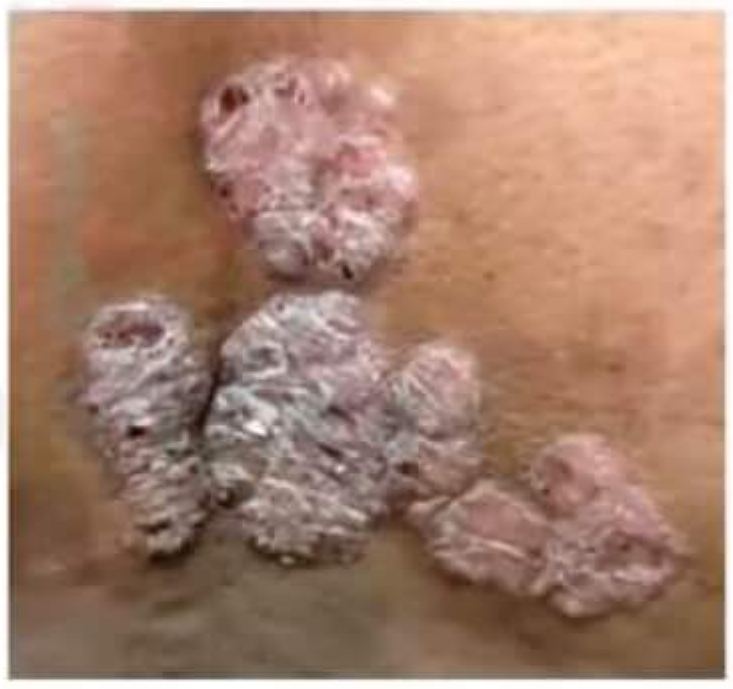


- Also known as cigarette paper like scales

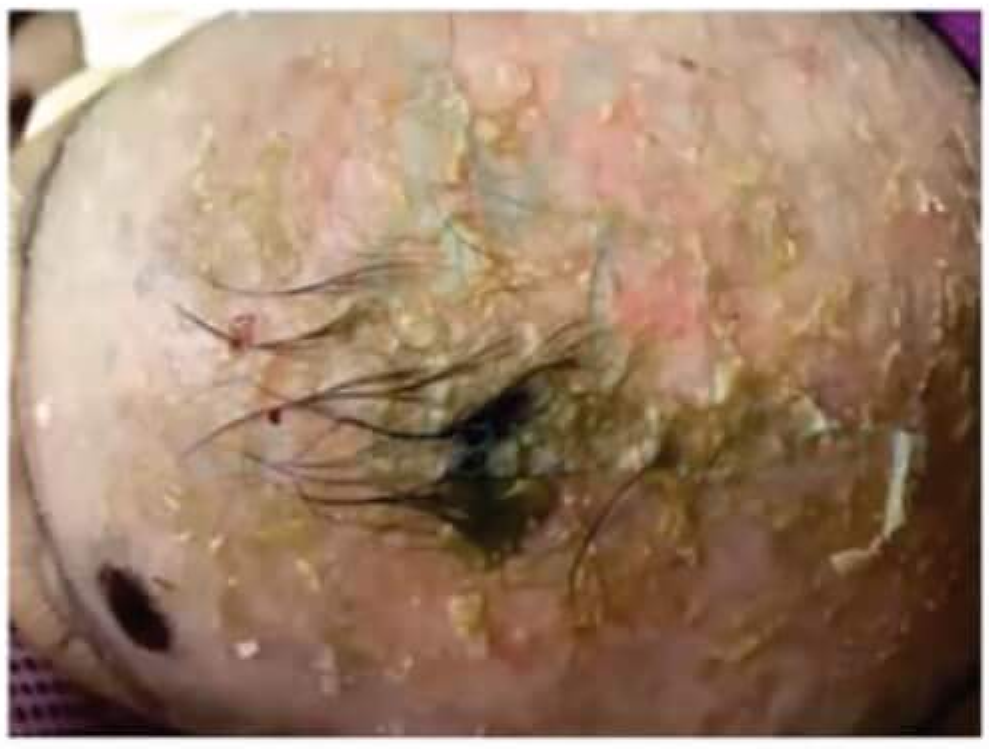
Oyster like (Heaped up) scales of rupioid psoriasis



Micaceous (silvery white)Q scales of psoriasis vulgaris

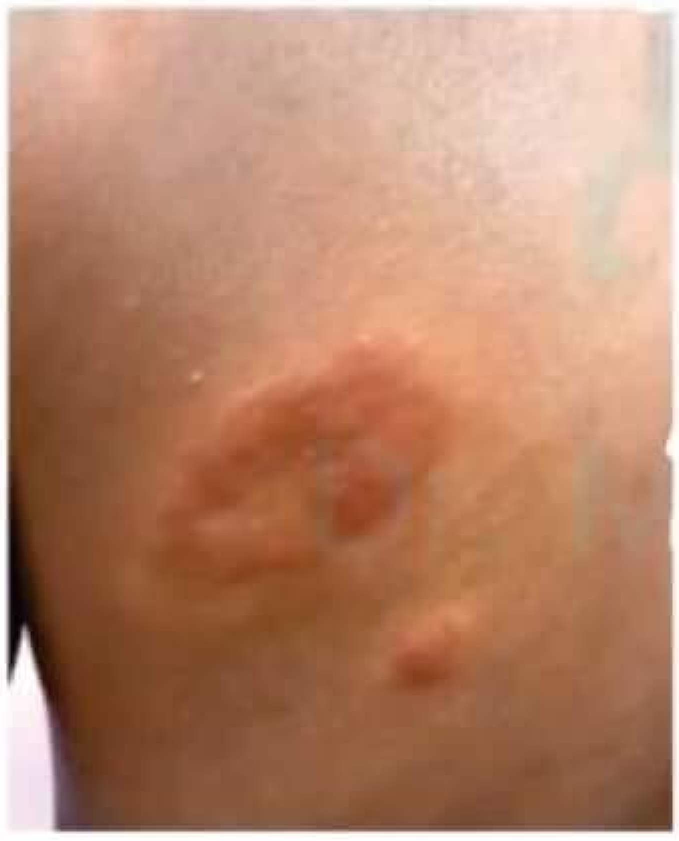


Greasy scales of seborrheic dermatitis in infant (also known as cradle capQ)

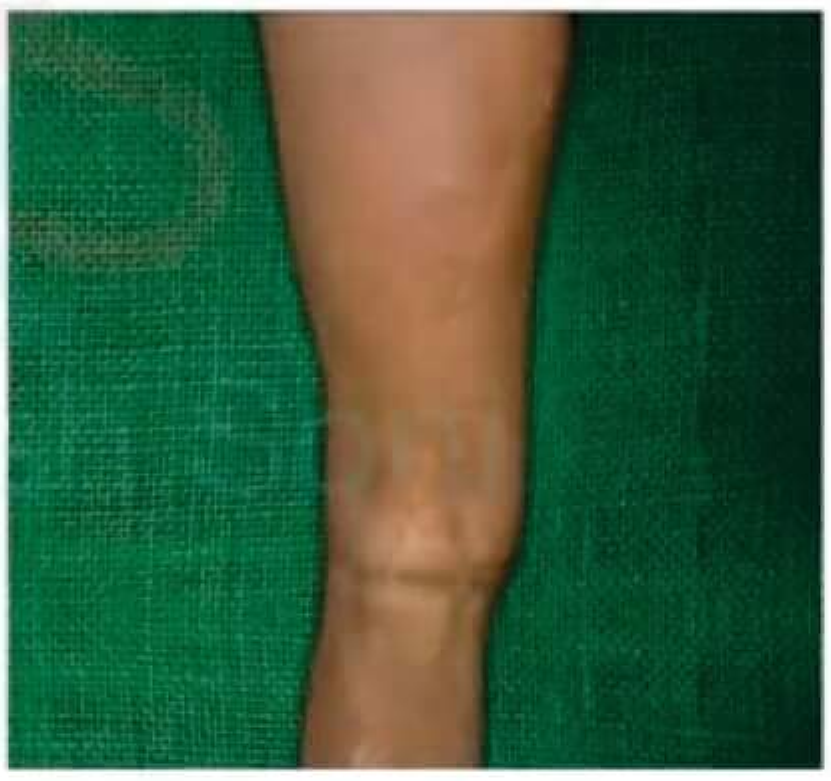


PATTERN AND ARRANGEMENT OF LESIONS

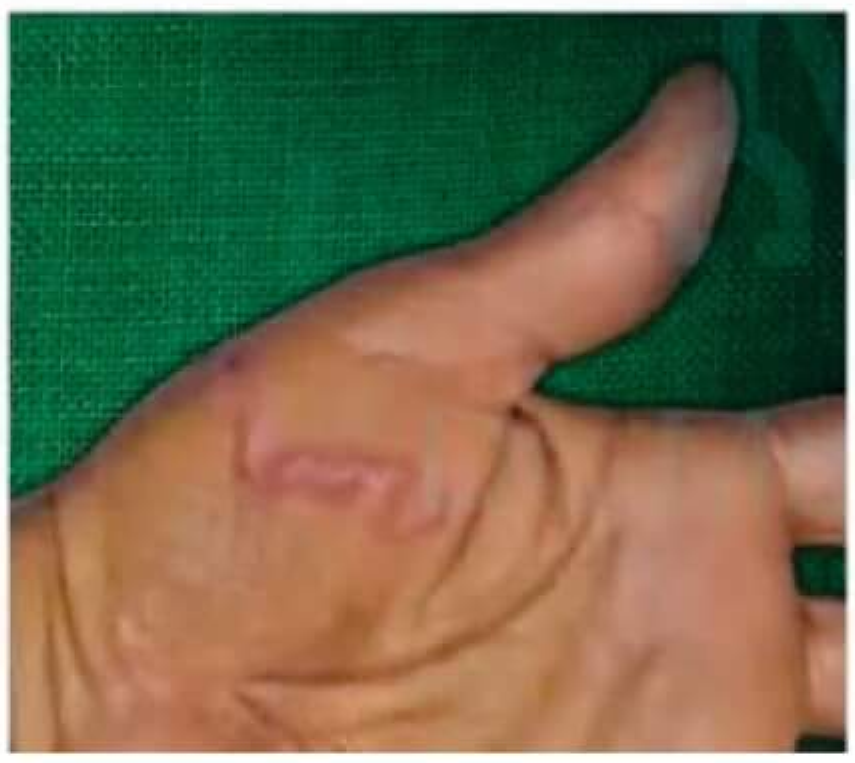
Annular lesion in a case of Hausen's disease (Leprosy)



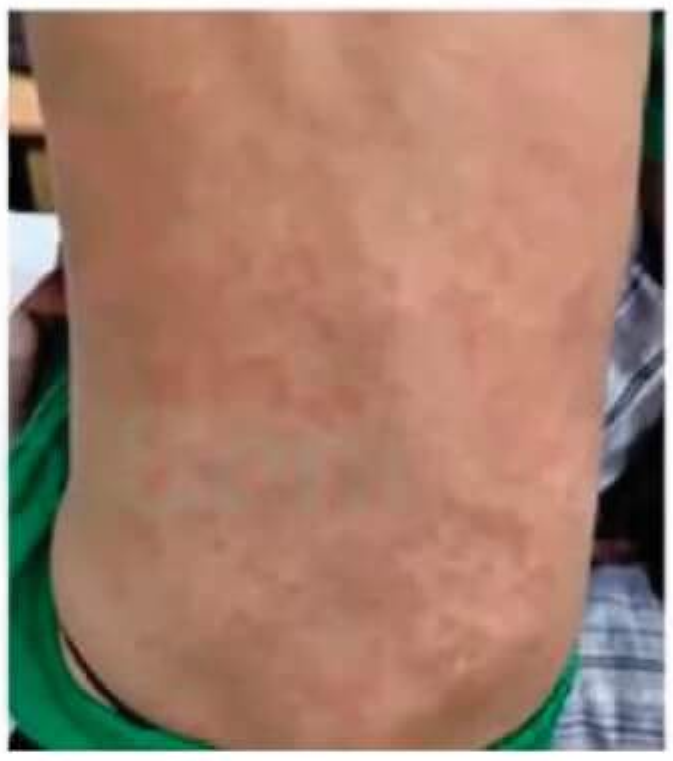
Linear lesions in a case of Lichen striatus



Serpiginous lesions seen in cutaneous larva migrans



Arcuate and polycyclic lesions in a case of urticaria

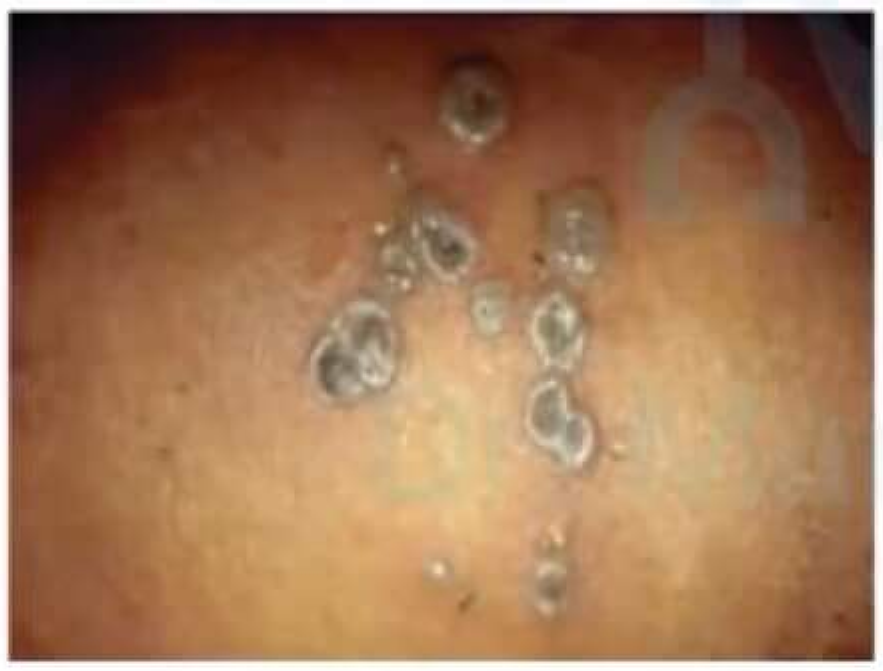


Multiple margins +

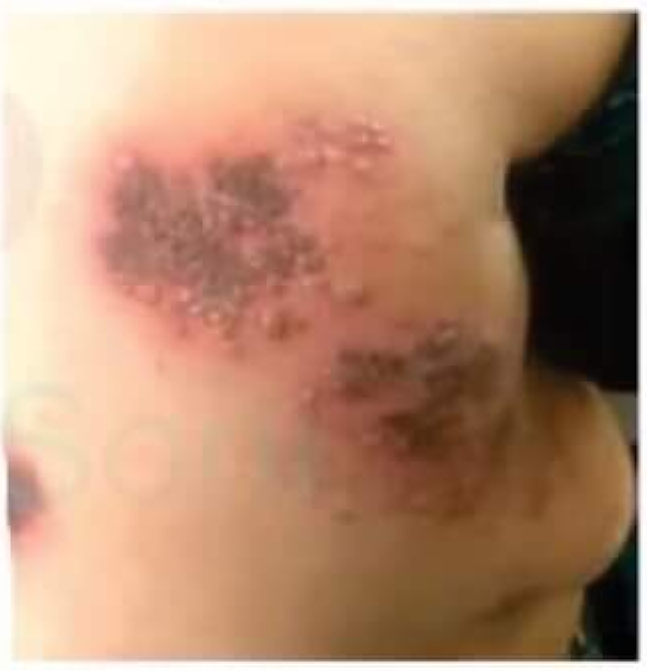
Target lesions Q – 3 concentric rings +

Arrangement and distribution of skin lesions

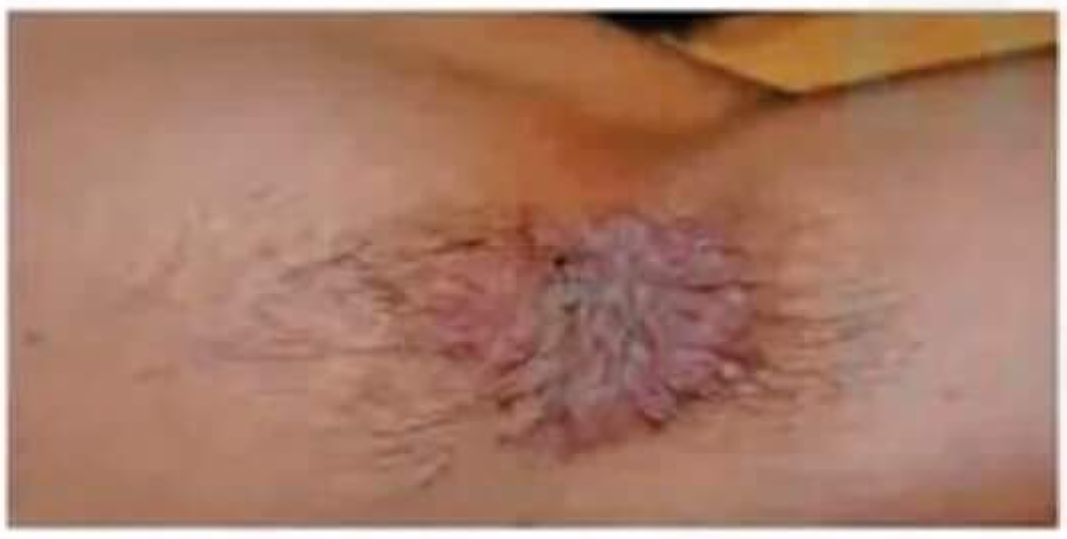
Herpetiform—clustered vesicles (fluid filled lesions);



Zosteriform—unilateral, dermatomal lesions of herpes zoster



Intertriginous (skin folds) involvement in Hailey-Hailey disease



Axilla

Acral distribution in a case of palmoplantar psoriasis